THE SURGICAL CLINICS OF NORTH AMERICA

NEW OPERATIONS

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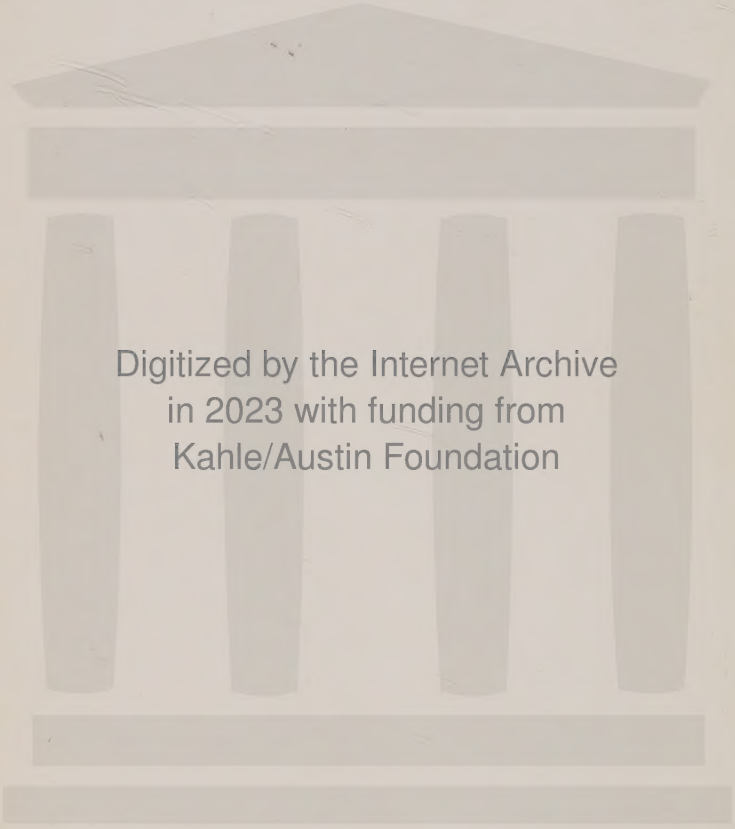
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THE SURGICAL CLINICS OF NORTH AMERICA

NEW OPERATIONS

PETER V. MOULDER, M.D., *Guest Editor*

Volume 46 — Number 1

FEBRUARY, 1966

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December 1966—*from Ochsner Clinic*

Thoracic Surgery
Alton Ochsner, *Guest Editor*

Table of Contents

SYMPOSIUM ON NEW OPERATIONS

FOREWORD 1
Peter V. Moulder, M.D., GUEST EDITOR

PERCUTANEOUS CORDOTOMY FOR PAIN 3
Sean Mullan, M.D.

HUMAN RENAL TRANSPLANTATION 13
John J. Bergan, M.D.; Vincent J. O'Conor, Jr., M.D.; John T. Grayhack, M.D.

PULMONARY EMBOLECTOMY 27
George R. Daicoff, M.D.; James J. Rams, M.D.; Peter V. Moulder, M.D.

THE TECHNIQUE OF EMERGENCY PORTACAVAL SHUNT 37
Frederick W. Preston, M.D.; Otto H. Trippel, M.D.

INTERNAL INSTRUMENTATION IN THE TREATMENT OF SCOLIOSIS 55
Ronald L. DeWald, M.D.; Claude N. Lambert, M.D.; Robert D. Ray, M.D., Ph.D.

PRESENT STATUS OF BILIARY TRACT SURGERY 69
John M. Beal, M.D.

A TECHNIQUE FOR DISSECTION OF THE AXILLA 77
George E. Block, M.D.; Sherwyn Warren, M.D.

OPERATION FOR OBSTRUCTING DUODENAL HEMATOMA 85
Robert J. Freeark, M.D.; William J. Norcross, M.D.; Richard D. Corley, M.D.

RECENT TRENDS IN SURGERY OF THE LARYNX 99
Stanton A. Friedberg, M.D.

MICROSURGERY OF THE EAR 111
John R. Lindsay, M.D.; George H. Conner, M.D.

A NEW TECHNIQUE EMPLOYING PARTIAL TEMPORAL BONE RESECTION IN PAROTID GLAND SURGERY FOR CANCER 131
George W. Allen, M.D.

PREOPERATIVE IRRADIATION AND BRONCHOPULMONARY SLEEVE RESECTION FOR LUNG CANCER 145
Robert J. Jensik, M.D.

SURGERY OF ACQUIRED DISEASE OF THE SMALL ARTERIES 161
James A. Hunter, M.D.; Hushang Javid, M.D., Ph.D.

NEW OPERATIONS IN CHILDREN'S CARDIAC DISEASE 175
James J. Rams, M.D.; George R. Daicoff, M.D.; Donald E. Cassels, M.D.; Peter V. Moulder, M.D.

SURGERY OF ACQUIRED HEART DISEASE 189
Peter V. Moulder, M.D.

VENA CAVA OPERATIONS FOR PREVENTION OF PULMONARY EMBOLISM 195
John J. Bergan, M.D.; Otto H. Trippel, M.D.

SURGICAL USES OF THE HYPERBARIC OXYGEN CHAMBER 209
Otto H. Trippel, M.D.; Michel N. Jurayj, M.D.; Charles J. Staley, M.D.; Jack van Elk, M.D.

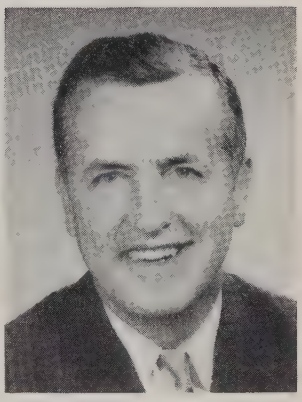
ADDITIONAL ARTICLE

Technique of Single-Layer End-to-End Intestinal Anastomosis by Triangulation 223
Carl J. Heifetz, M.D.

Index 229

SYMPOSIUM ON NEW OPERATIONS

Foreword



PETER V. MOULDER, M.D.

This symposium presents new operations and improved operative procedures currently employed in the Chicago area. One must remember that a truly new operation is rare, and even it will be based upon old surgical methods and skills. Most of the "new" operations represent improvements upon and adaptations of older operations or new applications of these based upon increased knowledge of pathophysiological processes. One is reminded of the story of the maestro who repeatedly nodded his head listening to a new symphony. Asked why he did this, he responded, "It is a beautiful symphony, I was merely bowing my head when I heard something from the great masters."

The operations described in the first three papers are truly spectacular. The effectiveness of Sean Mullan's simple new operations for the relief of pain has been amply demonstrated. Bergan and his colleagues comment on and relate personal experience in the homotransplantation of kidneys. Daicoff and his co-workers describe the technique of pulmonary embolectomy, dramatic when it saves a young patient, but disappointing from an overall viewpoint when it is seen that the number of cases in which the operation has been used remains small; nevertheless it has been a great stimulus to needed study of the whole problem of thromboembolism.

Portacaval shunt is an established operation, but the Preston-Trippel technique for emergency application has produced remarkable salvage in a dismal group of patients and thus claims newness for good reason.

DeWald's group has written a personal chronicle on the use of internal fixation techniques for scoliosis which clearly puts the problem in focus.

One of Chicago's new surgery chairmen, John Beal, describes new advances in biliary tract surgery, and his review is followed by descriptions of two neat operative techniques, the first by Block and Warren for axillary dissection and the second by Freeark and colleagues for the surgical treatment of the uncommon but increasing trauma problem, obstructing duodenal hematoma.

The otorhinolaryngologists (ENT men) have risen from near abolition of their practice by penicillin to present us with some fascinating surgery. Please note that they are putting foreign bodies intrepidly in sites which would make vascular surgeons shudder.

Jensik destroys another great worry of surgeons who have sidestepped operating in irradiated tissues by performing sleeve resections and hazardous bronchial anastomoses in patients with primary carcinoma of the lung who have had preoperative irradiation.

In the vascular and cardiac surgical fields most operations are constantly changing. Many modest new operations have been introduced and new applications made of older operations. Resort is made, therefore, to summaries rather than to many reports to give appropriate coverage to what is new in these areas. We are especially fortunate to have the benefit of the diverse experience of Hunter and Javid in arterial reconstruction.

Trippel and colleagues have contributed a description of their experience in and a candid evaluation of the use of hyperbaric oxygenation for surgical problems.

This symposium makes no pretense of including all of the new work in the area. Heavy schedules have prevented some men from contributing. To those who found it possible to share their talents so generously I am deeply grateful.

PETER V. MOULDER, M.D.
Guest Editor

Percutaneous Cordotomy for Pain

SEAN MULLAN, M.D., F.A.C.S., F.R.C.S. (Eng.)*

Medical practice consists in preventing and curing disease and in relieving the symptoms of those diseases which cannot be cured. A great deal of effort is spent, in fact, in relieving symptoms. This is especially true in the field of cancer therapy where the most insistent symptom, apart from the reactive depression, is perhaps pain. Other forms of pain no less intractable are encountered—the causalgias of nerve injury, the neuralgia of herpes zoster, the pain of amputations, of tabes, of chronic ischemia of the limb, of diabetic neuropathy and of osteoarthritis. For more than 50 years it has been possible to relieve many of these pains by the surgical operation of anterolateral cordotomy, yet when compared with the number of pain problems which present, this operation has been relatively little used. The reasons are many. It is a major operation. It has a significant mortality especially in the debilitated, there is post-operative pain, there is a period of hospitalization of at least ten days and a period of convalescence of at least three weeks. It is not always successful. Occasionally there is weakness, occasionally there is sphincter impairment. Frequently, sensation and pain return within a few years. In short, the hazards and uncertainties are such that it is reserved for the more truly desperate pains. The alternative treatment for serious pain is narcotic medication and it is, in many instances, even less desirable. Tolerance and addiction are so well established by six months, as a rule, that pain relief is obtained only by somniferous dosage. At the same time large doses are needed to satisfy addiction while the personality deteriorates under their continued use. Narcotics are inadequate for patients who have more than three or six months to live.

From the Division of Neurological Surgery, University of Chicago Hospitals and Clinics, Chicago, Illinois

Aided by grants from the USPHS General Research Support Grant 1-501-FR-05367-01 and USPHS Med. Res. NB-05445-01.

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The mortality and discomforts of classical surgical cordotomy depend largely upon the general anesthetic and laminectomy (or laminotomy) that are necessary in order to make a simple 5-mm. incision in the cord. These are often too much for the terminally ill patient who needs pain relief most of all. It has recently become possible to eliminate these difficulties by a simple percutaneous needle procedure done under local anesthesia. Although the vertebral laminae are closely packed together throughout most of the spinal column, there is a wide interval between the first and second cervical vertebrae in order to allow rotatory movements of the skull upon the neck. Here there is wide needle access to the posterior, lateral and even to the anterior surface of the cord. In our first series of percutaneous cordotomies we used a radioactive needle which lay against the anterolateral surface of the cord and delivered a radioactive necrotic dose to a depth of 5 mm. Later we used an electric needle which pierced the anterolateral quadrant of the cord and delivered a direct current (d.c.). Finally Dr. Rosomoff has used a similar electric needle but has used a radio-frequency current (r.f.). These three techniques will be described.

Percutaneous Strontium Cordotomy [1, 2]

The needle or rather the stilet of the needle, which was designed and made by Dr. Paul Harper, contains 54 millicuries of strontium-90 in its terminal 6 millimeters. Strontium-90, which has a half life of 28 years, decays to yttrium-90 with emission of a weak beta ray; yttrium-90, which has a half life of 2.8 days, decays to zirconium-90 with emission of a strong beta ray. Thus the stilet, which is a miniature nuclear reactor, has a long half life and a strong penetrating emission. A 15-minute period of radiation delivers 16,000 rads at a distance of 3 mm., 6000 rads at 4 mm., 2000 rads at 5 mm., and 0 rads at 9.5 mm. A five-minute dose delivers 2000 rads at 4 mm. and a 60 minute dose delivers 2000 at 6 mm. It is usually assumed that 2000 rads when delivered in one dose are necrotic to neural tissue.

A local anesthetic, lidocaine (Xylocaine) 1 per cent or procaine 1 per cent, is used. The patient may sit upright or lie prone. A thin-wall No. 17 needle carrying an inactive stilet is inserted from a skin point midway between the mastoid processes and the posterior midline interval between C1 and C2 in a direction at 45 degrees to the cord. When it pierces the posterolateral dura the stilet is advanced lateral to the cord until it touches the inner aspect of the anterior dura at a point 3 mm. short of the midline of the odontoid process. Control of the needle is effected by multiple biplane rapidly developing radiographs which relate its position to bony landmarks at each stage of advancement. Correction of the

position of the needle point can be made by applying traction to the handle of the needle by means of adhesive tape affixed to the skin of the face or neck or to some external object. When a satisfactory position is achieved, the inactive stilet is replaced by the strontium stilet and this remains in place for 15 minutes. The total procedure takes about 30 minutes to perform.

The patient experiences no discomfort during irradiation and may walk back to his room or even leave the hospital on the same day. Since the No. 17 needle leaves a moderate size puncture in the dura which allows cerebrospinal fluid to leak into the muscle tissue, it is best for the patient to lie down for several hours to avoid a lumbar puncture type of headache. The site of needle puncture may ache for a few days.

Relief of pain begins within two or three days as a rule and generally this occurs a day or more before sensory loss is apparent. This suggests that fibers carrying some of the patient's pain are more sensitive to irradiation then those carrying pin prick. By the end of the week, pin-prick loss has usually reached the groin and by one to three months it has reached the level of the second cervical dermatome.

Thus the procedure is a simple one to perform—not much more difficult for the patient than a lumbar puncture—and gives good early relief of pain in the lower half of the body. Relief of pain in the upper half of the body is too slow to be satisfactory in most instances. Early relief can be provided by 30-minute irradiation in patients whose life span is less than three months. Such a lesion could invade the pyramidal tract and cause ipsilateral limb weakness after about three months in those of longer life span.

Percutaneous Direct Current Intramedullary Cordotomy[3]

This procedure takes about two hours to perform and therefore demands more tolerance on the part of the patient though it is not more painful. Local anesthesia is used. The patient lies supine. A No. 22 needle is inserted horizontally under biplane x-ray guidance from a skin point 1 cm. below and 1 cm. behind the tip of the mastoid process (Fig. 1). The dura and arachnoid are pierced anterior to the dentate ligament. This penetration usually causes a slight flick of pain. A drop of fluid will appear at the end of the needle. Ten cubic centimeters of air and 0.25 cc. of iophendylate (Pantopaque) are injected into the subarachnoid space without withdrawing fluid (Fig. 2). Care should be taken that the patient's head is arched very slightly backward so that air does not enter the skull to cause headache. At the same time it must not be arched too far backward because the Pantopaque, instead of sitting on the dentate ligament, will fall into the cisterna magnum. Too much Pantopaque must not be

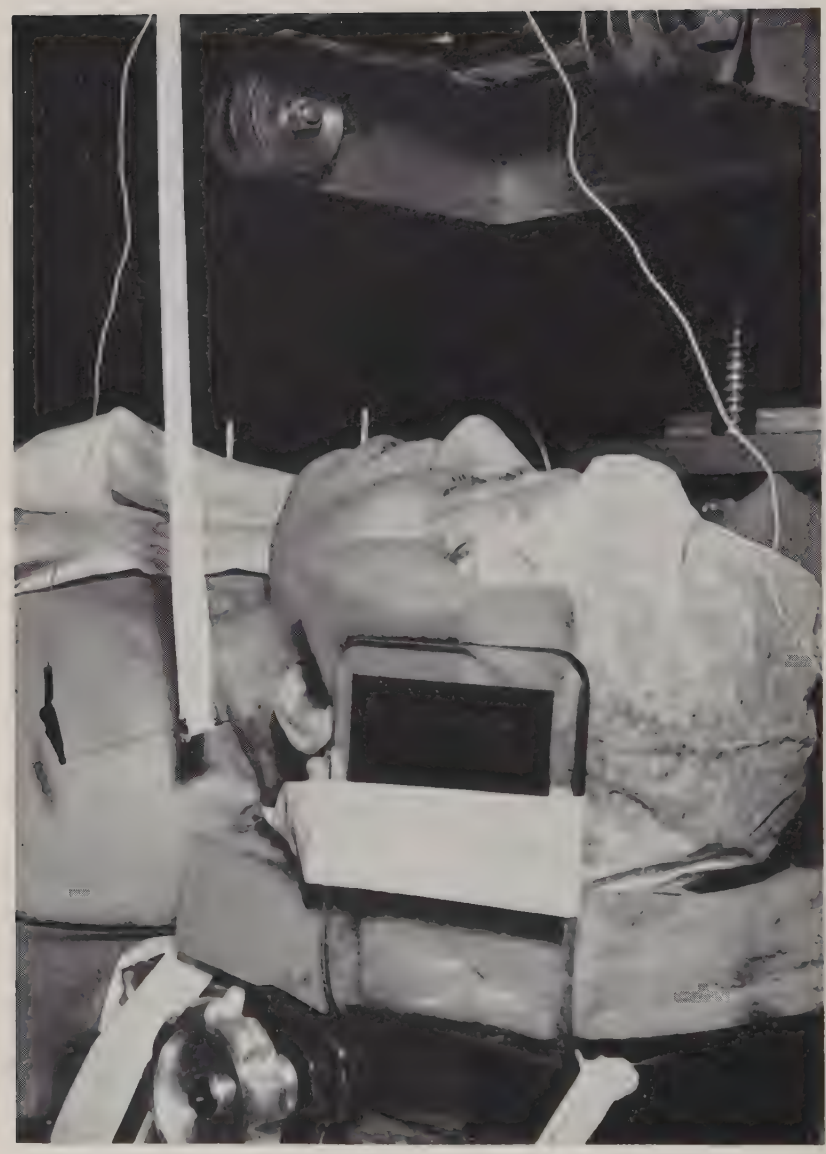

Figure 1. This is the extent of the operative procedure for percutaneous direct current intramedullary cordotomy. The patient's head rests comfortably in a sponge lined U-shaped head holder. A No. 22 needle has been inserted below the mastoid process. An electrode has been inserted through it into the upper cervical cord. The position of the needle is controlled by biplane radiographic technique using small polaroid film.

used because it will pool over the odontoid process and obscure its midline (which is taken to be the midline of the cord).*

The point of insertion of the electrode into the cord is 2 to 3 mm. in front of the dentate ligament. If the directing needle is too far forward or backward, it can be corrected to about 2 to 3 mm. by angling the external end of the needle and anchoring it by tape. A more extensive correction requires withdrawal and repositioning of the needle. An electrode ten thousandths of an inch in diameter made of platinum (70 per cent) and iridium (30 per cent) alloy, insulated except for its terminal 2 mm., is inserted into the outer aspect of the cord if leg analgesia is required or into its medial aspect if arm analgesia is desired. Penetration of the pia causes slight pain in most instances. A single direct current (approximately 0.5 milliampere, 4 volts) stimulus is applied. It will cause an ipsilateral jerk of the arm and leg if it is too near the pyramidal tract. It will cause an ipsilateral jerk of the neck if it is in the anterior horn or in the anterior horn fibers. Ideally, it will cause a tingling or sensation of warmth or cold (but not of touch or pain) in the area in which analgesia is desired. If it produces these sensations in an adjoining area, then the needle may be directed medially or laterally as necessary. Often stimulation produces no sensation although subsequent events demonstrate that the placement was correct. In these situations reliance must be placed upon anatomical landmarks. Sometimes when an increasing direct

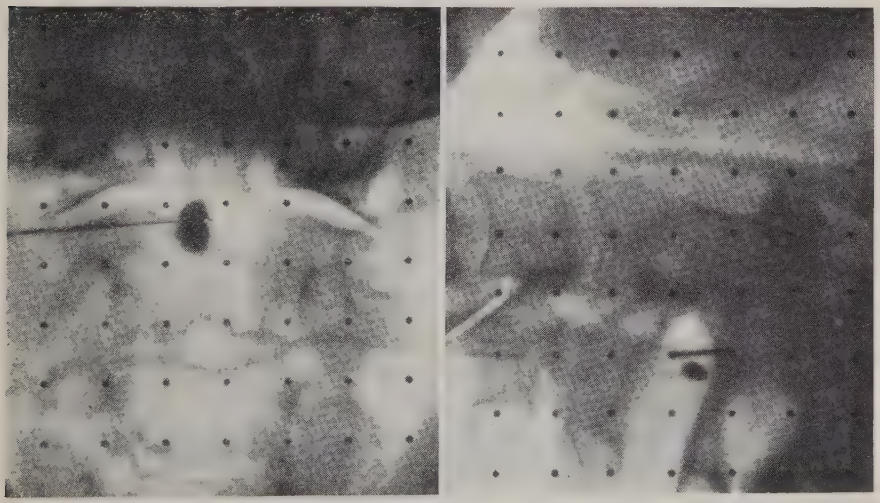

Figure 2. Anteroposterior view of upper cervical spine (through the mouth) shows Pantopaque globule sitting on dentate ligament. The percutaneous needle pierces the cord almost to the midline. *Right,* Lateral view shows point of needle lying between the dentate (outlined by Pantopaque globule) and the anterior surface of the cord (outlined by air).

* Since submission of this article we have used 1 cc. of iophendylate (Pantopaque) emulsified (by shaking) with 1 cc. of cerebrospinal fluid. This gives a superior picture.

current stimulus fails to elicit a response, an alternating current stimulus will prove satisfactory.

Either the anode or cathode may be used to produce a lesion. The indifferent electrode consists of a plate applied to the skin of the adjoining shoulder. It should be remembered that a cathode lesion produces nearly twice as large a lesion as the anode. The current employed is usually 1000 microamperes at a voltage of about 4. As anode lesions are occasionally associated with some pain in the neck during production of the lesion, we have lately preferred cathode lesions. This allows us to use a lower current.

After five to ten minutes, analgesia begins to develop and usually is more dense in one part of the body than another. If, for example, it is more dense in the trunk than in the arm and arm analgesia is required, then, as soon as this difference is detected, the electrode is moved more medially. A good lesion usually develops within 15 minutes with the cathode or within 30 minutes with the anode. If a lesion does not develop the needle may be too far anteriorly. While the lesion progresses, motor as well as sensory function is constantly examined. At any signs of motor dysfunction, the lesion is stopped. If the sensory lesion is inadequate the electrode is reinserted more anteriorly. These direct current lesions tend to regress after the current is stopped and, for this reason, we usually keep the current going for ten minutes after a satisfactory level has been reached in an attempt to insure greater permanency of the lesion.

When the lesion is completed the patient usually feels tired and when he stands there may be a lack of tone in the ipsilateral leg even though there is absolutely no abnormal pyramidal sign. This hypotonia lasts a few days and resembles that usually found immediately after a standard surgical cordotomy but it is less intense. These patients walk about on the same day but usually prefer to wait a day in the hospital before going home. The site of needle puncture may ache for a few days.

Percutaneous Intramedullary Radio-frequency Cordotomy

We use exactly the same needle-inserting technique as for the direct current cordotomy, but use a needle with a 3-mm. uninsulated tip. The radio-frequency current produces a lesion by heat coagulation. We use a setting of 100 milliamperes (Codman and Shurtleff machine) and begin with a five-second lesion. This does not usually produce any clinical results. For each subsequent application the current is increased by increments of five seconds until the desired sensory loss is produced, usually between 30 and 60 seconds. During each application of the current, motor power is tested continuously. The current could be stopped at any sign of motor weakness. The radio-frequency lesion causes quite a significant pain in the neck as the critical temperature is reached. A lesion then

appears very suddenly and involves the total contralateral side of the body below C2. We have not been able to achieve a selective analgesia of arm or trunk. As with the direct current lesion there is an ipsilateral hypotonia in the leg without demonstrable pyramidal signs. In several patients we have noted that, on the following day or days, there is a significant weakness in the ipsilateral leg which, however, always disappears. This suggests that a significant degree of swelling develops about the lesion within the next few days.

DISCUSSION

We have now performed 266 percutaneous cordotomies for 200 patients. There have been 148 strontium lesions for 99 patients, 99 direct current lesions for 84 patients, and 13 radio-frequency lesions for 11 patients. For our last six patients we have developed a lesion by means of the direct current and then applied the radio-frequency current before withdrawing the needle. The excess of cordotomies over patients is due to the fact that some patients have had bilateral cordotomies and some have had repeat procedures because the first one failed. This was particularly true in the early cases.

The strontium procedure is the easiest to perform. It gives excellent early relief of pain to patients with pain in the lower half of the body due to cancer. It is the preferred method for patients of this group who cannot lie quietly for the two hours demanded by the direct current procedure. We do not recommend it for pain in the chest or arm because sensory loss and pain relief are delayed. We do not recommend it for patients with a normal life span because we do not yet know the late effect of irradiation of several hundred rads which penetrate the pyramidal tracts beyond the limits of our deliberate necrosis.

Percutaneous direct current cordotomy is the most satisfactory to perform because the lesion develops very slowly over a period of 10 to 30 minutes while the patient is still on the table. This gives ample time to alter the position of the needle and the strength of the current to insure optimum results. It has one real disadvantage, and that is the tendency for the sensory level to drop in those patients followed for a period of greater than six months. Because of its simplicity, however, the lesion may be repeated if the sensory level falls. For patients with pain in the upper half of the body with a life expectancy of less than six months, this certainly seems to be the most suitable procedure. Until we have more experience, the radio-frequency lesion is probably the preferred lesion for all groups. It may be that the creation of larger lesions by the cathode current will solve the problem of late shrinkage of the analgesic

zone. A cordotomy with an assured shrinkage is perhaps an asset and its role is being investigated in the treatment of herpes zoster and other nonmalignant conditions.

Although our own follow-up of percutaneous radio-frequency cordotomies is not yet long enough, Rosomoff's series shows that the sensory level holds up well to six months. A 12-month follow-up is not yet available. The disadvantages of this procedure are the pain during development of the lesion, the suddenness of the development of the lesion giving little time for correction should the needle be wrongly placed, and the inability to produce a selective arm or trunk lesion. The reactive swelling which occurs within the cord during the next few days is particularly significant when the second stage of a bilateral lesion is performed. It is probable that refinements will improve the method.

We have tended to avoid bilateral cordotomies when possible because of their recognized hazards by the classical surgical method and because, in both our strontium and direct current series, paralysis of the bladder has occurred in 50 per cent of those patients who had intact sphincters before the procedure (three out of six patients in each series). Rosomoff has had much less trouble with his radio-frequency series. It should be noted that his technique varies slightly in that he uses a larger needle (No. 18) with a steel electrode and inserts it more anteriorly in the cord.[4]

We are also concerned about bilateral cordotomies because of the risk to life. We have lost one patient in the direct current and one in the radio-frequency series after the second stage. Both patients were perfectly well after the procedure but expired suddenly and unexpectly within 24 hours. It is our belief that swelling within the cord interfered with either respiratory or vasomotor control. For this reason we are inclined to favor the strontium procedure for the second side. We have had no mortality in the strontium series and detected no undesirable cord swelling (the lesion develops slowly).

Our failures have been due mainly to inadequate selection. They fall into three groups. Some patients with terminal cancer who have complained bitterly of their preoperative pain have continued to complain no less bitterly of anorexia, weight loss, poor sleep or pain elsewhere in the body postoperatively. In other words, their reactive depression was more important than their pain and this depression, of course, was not altered. Patients already addicted to narcotics have seldom given up their narcotics even when the painful area became analgesic to pin prick. A few patients, owing to extreme nervousness, could not lie still long enough to allow completion of the procedure with safety, and it had to be abandoned. There is another small but puzzling group of patients who have continued to complain of deep pain and of pain on deep pressure even though the corresponding skin area was completely analgesic. This

has caused us to consider the possibility that some portions of deep pain may ascend in the ipsilateral side of the cord in some patients.

The main advantages of percutaneous cordotomy lie in the technical simplicity and ease for the patient. It has thus been possible to provide cordotomy relief to a vast number of terminally ill patients who could not possibly be expected to survive a surgical cordotomy. It has also been offered to patients whose pains were not yet severe enough to warrant a major operation and to patients with nonmalignant disease which was not serious enough to justify the risks of the classical procedure. It has proved so satisfactory to patients and to their referring physicians that the number of cordotomies we have performed has increased more than fifteen fold over the number performed by the classical surgical procedure in the previous comparable period. Weakness (as distinct from hypotonia) as a complication has virtually disappeared with increasing experience. A mortality factor of less than 1 per cent compares very favorably with the standard surgical mortality factor of between four and 25 per cent, especially since most of our patients were in a very debilitated state. With the use of the strontium lesion for the second stage or a greater interval between the stages of bilateral procedures, we believe that the mortality factor could be eliminated.

Technically the procedure is still relatively new and undoubtly further improvements will occur. A very significant advance has been the use of rapidly developing 4 by 5 inch Polaroid radiographs.* Type 57, with a speed of 3000, has given adequate but not exceptionally good pictures. The new TLX film in a specially designed cassette developer gives pictures of excellent quality. Television fluoroscopic image intensification has been used by us and by Rosomoff and provides good radiographic control but the rather complicated equipment that is required probably makes it impracticable for general use. We have also developed and used an x-ray controlled instrument which guides the needle insertion mechanically. This facilitates the procedure but, since manual insertion is a fairly simple technique, the use of this special equipment is not essential except for investigative work. The use of a nitrogen-cooled needle has been considered but found unsuitable because practical engineering factors require a needle which seems to be too large for insertion into the spinal cord. A fiber optic conducted laser beam has also been investigated but it, too, has been unsuitable. Perhaps the most useful, immediately available refinement will be a radio-frequency electrode of fine diameter that records temperature at its tip, or a flexible electrode that will emerge eccentrically from the introducing needle.

* Polaroid Radiographic Packets, special x-ray cassettes and processing equipment were supplied through the courtesy of Polaroid Corporation, X-ray Technical Service Department.

SUMMARY

1. Interruption of the pain-conducting fibers in the anterolateral tract of the spinal cord may be accomplished by a simple percutaneous needle puncture under local anesthesia.

2. Three techniques are described: (1) percutaneous radioactive strontium cordotomy by which the cord is irradiated to a depth of 5 mm. from an intradurally inserted strontium needle; (2) percutaneous electric direct current cordotomy; and (3) percutaneous electric radio-frequency cordotomy. In the latter two the lesion is made in the cord by means of an electrode inserted into the cord substance.

3. These simple techniques have eliminated so much of the inconvenience, mortality and morbidity of the classical surgical anterolateral cordotomy that the benefits of cordotomy may now be extended to a great many patients to whom they were formerly denied, especially those with terminal cancer.

REFERENCES

1. Mullan, S., Harper, P. V., Tani, E., Vailati, G. and Lathrop, K. A.: A nuclear needle for use in neurosurgery. J. Neurosurg. *20:* 940–947, 1963.
2. Mullan, S., Harper, P. V., Hekmatpanah, J., Torres, H. and Dobben, G.: Percutaneous interruption of spinal-pain tracts by means of a strontium-90 needle. J. Neurosurg. *20:* 931–939, 1963.
3. Mullan, S.: New technics in neurosurgery. Postgrad. Med. *37:* 636–641, 1965.
4. Rosomoff, H. L.: Personal communication.

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Human Renal Transplantation

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"Clinical transplantation is itself an experimental procedure, but that is no longer just a polite way of saying that the transplantation of kidneys is more often than not a failure."

P. B. MEDAWAR

Transplantation of a kidney from one person to another is a form of treatment for uremia. In certain circumstances it is an effective method of treatment. Nevertheless, despite well publicized recent advances, clinical renal transplantation is still an experimental procedure, not a therapeutic one.

Since human allografting is purely an experiment it is at present conducted in an environment acceptable to the study of method, collection of data, assessment of results, analysis and criticism of technique, and manipulation of variables. Thus advances in this infant clinical science have been marked by a rapid interchange of information between investigators, by international collection and dissemination of data, and by withering self-criticism of accomplishments. Not all of the criticism of these efforts has come from within. Powerful and influential voices

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have indicated the physical, financial and emotional suffering which transplantation programs have caused.[10]

The multidisciplinary needs of management of the transplant patient have tended to humble each contributor. Interdependence of each of the many medical sciences involved has served to stimulate a variety of approaches to the many problems and to strengthen each specialty.

It is appropriate to discuss renal transplantation in this volume which is devoted to new surgical operations, not to suggest that renal transplantation be expanded into many clinical centers and community hospitals but rather that the operation be examined to assess its current status and possible future. Thus, it may be possible to assess the eventual role of renal grafting as a possible therapeutic procedure.

Human renal transplantation in the modern period has passed through several necessary stages. These include the experience gained with unmodified recipients described by Hume;[9] the identical twin contribution of the Harvard group at the Peter Bent Brigham Hospital;[19] the nonidentical twin explorations contributed by Hamburger and other French workers; the immune suppression with total body irradiation so magnificently performed also by Hamburger;[8] and the development of azathioprine (Imuran) by the Burroughs Wellcome Company.[4] This latter contribution has allowed effective immune suppression. From this has come the realization that a spectrum of histocompatibility differences exists between individuals and that rejection of organ grafts is not an all-or-none phenomenon. In fact, experience has shown that attenuation of rejection can lead to a state of relative host-graft nonreactivity. Another by-product of this experience of attenuation of maximal rejection has been the reemphasis upon the need for clearly defining and identifying genetic tissue differences between individuals. In short, it has emphasized the need for tissue typing.

Following development of Imuran, a great stimulus was given to transplantation. Many centers expanded existing laboratory investigations. Immunologic studies began to assume a spirit of urgency, and many groups began clinical transplantation. As this occurred, it was obvious that a team approach to this problem was mandatory (Fig. 1). Each representative of the scientific disciplines contributed to care of the renal recipient and aided in education of the other team members regarding the possibilities and limitations of his own field. As the technical problems of the early postoperative period were overcome, later difficulties served to emphasize this need for a team approach. Late complications of infectious disease, for example, required special knowledge of management of patients with local and disseminated mycoses, local and systemic viral diseases such as herpes zoster, and overwhelming pulmonary infection with a diversity of organisms including cytomegalovirus and pneumo-

cystitis carinii.[20, 21] Other direct complications of therapy included aseptic bone necrosis secondary to corticosteroid administration, steroid diabetes, relative bone marrow aplasia and atypical connective-tissue diseases, all of which required specialized knowledge for effective treatment.[26]

The enormous amount of energy diverted into transplant programs was demonstrated by the fact that the groups currently active had performed 672 primary renal transplants by May, 1965. In addition, 45 secondary transplants had been done and two tertiary grafts were accomplished.[17] An estimate of some of the financial burden of this work was indicated by the Denver Veterans Administration Hospital which cited a cost of $272,773.00 for an 18-month program during which time 28 renal transplants, 3 liver grafts and 4 splenic allografts were done.[2]

OPERATIVE TECHNIQUE

Operations associated with transplantation of the human kidney consist of the transplant procedure itself with exposure, vascular anastomoses and establishment of urinary continuity; bilateral recipient nephrectomy performed as an integral part of the transplant procedure

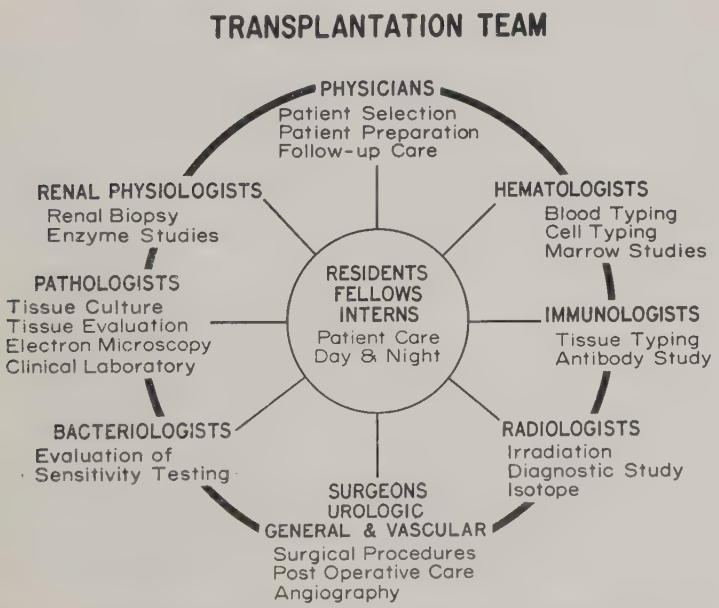

Figure 1. The transplantation team is a complex multidisciplinary group which is organized to give the highest quality medical care to the transplant donor and recipient while simultaneously obtaining and recording the most complete information possible in order to advance the science of transplantation.

or done before or after establishment of new kidney function; and ablation procedures such as thymectomy and splenectomy done with a view toward favoring graft acceptance.

The transplant operation as modified by the Harvard group from Kuss' original procedure has proved to be satisfactory in most instances.[18] Details of performance of this operation are also found in Calne's excellent monograph and in Starzl's monumental report of the Denver group's surgical experience.[22] Special problems arising in children are solved by modification of the basic techniques.[24]

The implant operation utilizes a lower abdominal extraperitoneal exposure of the iliac vessels and bladder so that the renal artery and vein can be connected to the hypogastric artery and iliac vein and the ureter or renal pelvis of the donor kidney can be anastomosed to the bladder or recipient ureter. If double renal arteries are encountered, hypogastric branch vessels are used in anastomosis.

Initiation of the transplantation operation of course begins with selection and preoperative management of the recipient. Suffice it to say that the recipient must be in end-stage uremia dependent for life upon peritoneal dialysis or hemodialysis. It is desirable but not essential that there be a family relationship between donor and recipient to minimize antigenic dissimilarities. The longest transplantation successes to date have been between closely related individuals. There must be ABO blood compatibility.[23] Absence of infection in the donor kidney and presence of a normal lower urinary tract in the recipient are mandatory. Absence of infection in the recipient prior to institution of immunosuppressive therapy is an absolute necessity. The fact that an occasional report describes the flouting of any or all of these factors appears only to emphasize their general desirability.[13]

At the time of surgery, painstaking attention is paid to details which insure technical perfection. It is clear that in the renal transplant recipient whose already ravaged defense and healing mechanisms are further insulted by administration of potent antimetabolite drugs and massive doses of corticosteroids, attention to the principles of surgical technique is of urgent importance. The drugs to be used for immunosuppression interfere with protein synthesis, and defects in wound healing are to be expected. Sharp incision of tissues to expose structures is used throughout the operation, obsessive attention to hemostasis is carried out at all times, wound edges are carefully padded against retractor trauma, and rough handling of structures is avoided.

A useful incision to expose the iliac bifurcation for the implantation consists of an S-shaped oblique lower quadrant cutting of skin and subcutaneous tissues. The lateral transverse limb above the iliac spine allows adequate exposure of depths of the wound while the medial limb gives

excellent visualization of the bladder. Slitting the external oblique muscle in the direction of its fibers for the entire length of the skin incision is easily accomplished. It is then necessary to divide the underlying internal oblique and transversus abdominis muscles perpendicular to the direction of their fibers. This can be done near the edge of the rectus sheath in order to utilize the fibroaponeurotic nature of the muscular attachments for a stronger eventual closure. Once these muscles are divided, the peritoneum and its contents are reflected superiorly and medially in such a manner that the common iliac artery is widely exposed.

Wide mobilization of the artery and vein is practiced. Freeing the common iliac artery as well as the external iliac allows some rotation of these vessels when the hypogastric is used in an end-to-end connection with the renal artery. Thus acute angulation and kinking of the hypogastric can be avoided (Fig. 2).

The venous anastomosis is usually performed in an end-to-side fashion using the external iliac vein. It is more convenient to perform this anastomosis prior to connecting the arterial inflow since the iliac venous clamps can be applied, an ellipse of vein excised, and stay sutures placed before the kidney is received from the donor operating room. When the venous anastomosis is completed, a bulldog clamp across the

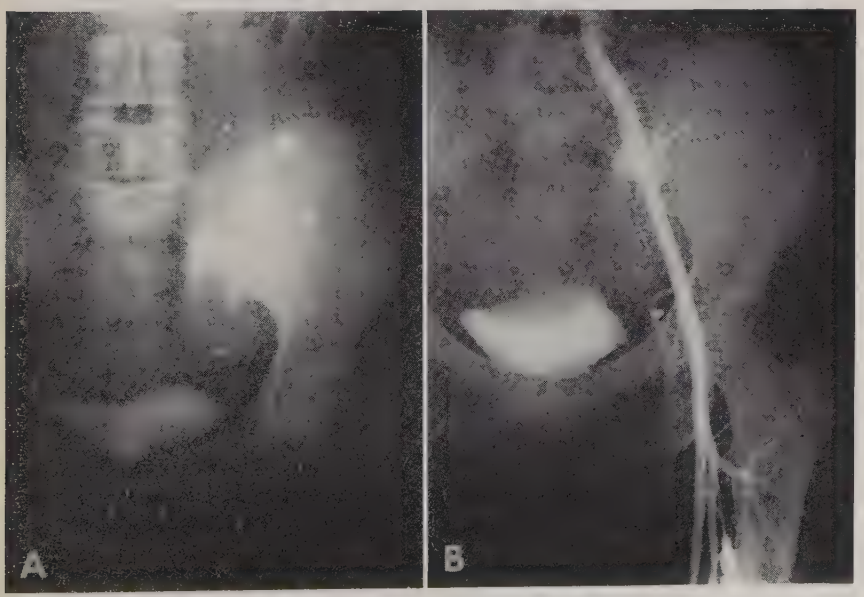

Figure 2. *A*, The urogram shows a well functioning transplant in the usual location in the left iliac fossa.

B, This arteriogram of the left iliac system shows the hypogastric to renal anastomosis as well as the intrarenal arborization within the graft. This sibling transplant demonstrated smoldering chronic rejection for one year before failing.

Figure 3. Phlebography of the right iliac system shows how little distortion is caused by an end-to-side renal vein to iliac vein anastomosis. The contrast media is diluted by renal venous effluent.

renal vein allows removal of the iliac occlusive clamps and restoration of venous flow. If a short renal vein is present, tension can be avoided by dividing the iliac vein and performing an end-to-end anastomosis of renal vein to proximal iliac vein. No distal venous stasis will result if prior thrombophlebitis has not been present (Fig. 3).

The arterial anastomosis is performed in an end-to-end fashion with conventional over-and-over continuous suture of fine Teflon-coated dacron. Spatulation of the ends of the vessels insures against stenosis of the anastomotic site. When precocious atherosclerosis of the hypogastric artery has destroyed its lumen, a simple end-to-side anastomosis of renal artery to the external iliac can be done. While Mims has suggested the hemodynamic unsuitability of this type of anastomosis, no functional disability has been noted by those using it.[16]

Since cooling the kidney has been shown to protect against ischemic tubular damage, it is advisable to utilize cold perfusion of this organ

especially when accompanying factors cast doubt upon viability or function of the graft, the recipient nephrectomy can be deferred. The advantage gained is insurance of excellent renal function at the time of the bilateral nephrectomy.

Splenectomy

Splenectomy has been performed in conjunction with bilateral recipient nephrectomy and renal transplantation in order to decrease total body lymphoid mass and to permit larger doses of immunosuppressive drugs during the usual postsplenectomy leukocytosis (Fig. 4). However, thromboembolic complications occur following splenectomy and Veith's work with Murray has discredited this portion of the transplant operation.[25] Veith showed that splenectomy neither prolonged graft acceptance nor allowed use of greater doses of immunosuppressive drugs. Splenectomy is not now regularly done as an adjunct to transplantation.

Thymectomy

The thymus is believed to be the original source of lymphoid elements concerned with immunologic reactivity. Proliferation of lymphocytes is chiefly a function of the thymus which is then the source of the circulating

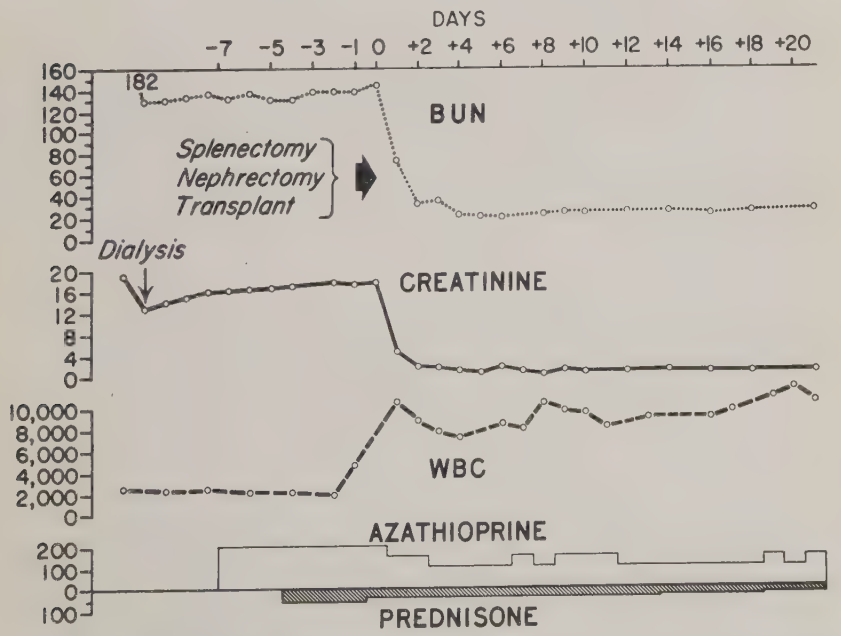

Figure 4. The pre-transplant preparation and early post-transplant progress is shown on this composite chart.

the ureteroneocystostomy can be done with deliberation. A submucosal tunnel 1.5 to 2.0 cm. in length is created with the mucosal opening lying near the trigone. The mucosal anastomosis is made without eversion and contains fine, irregularly spaced catgut sutures. The posterior bladder wall window is left patulous to avoid postoperative stricture and ureteral obstruction. Multiple layer, water-tight cystostomy closure is of importance because of the poor healing potential of the characteristic transplant patient.

The Northwestern group has utilized an inlying urethral catheter for five to seven days to afford total bladder decompression. In addition, placement of fine siliconized polyethylene suction drains has allowed perirenal drainage without the possibility of introduction of infection in retrograde direction. The suction drains are removed after the urethral catheter is taken out. This innovation in technique has proved its value as an adjunct to the transplant operation.

Recipient Nephrectomy

Bilateral nephrectomy in the transplant recipient is done to remove a potential source of infection, to ameliorate the hypertension which is stimulated by the diseased kidneys, or to reduce the hazard of transmitting the patient's original disease to the new substrate.

It is generally agreed that grossly infected kidneys should be removed before initiation of immunosuppressive therapy. When secure wound healing is achieved, preparations can be begun for transplantation. Such unanimity of opinion does not attend performing nephrectomy to prevent development of nephritis in the transplant. Although Murray has suggested that hazards of transmission of glomerulonephritis from the recipient to the transplant are lessened by nephrectomy before or during the implant, evidence for this is lacking. Indeed, changes resembling nephritis have been found in the transplant even when the patient's original disease has been pyelonephritis. Woodruff suggests that there is no obvious reason why nephrectomy should lessen the risk of nephritis developing in the transplanted kidney.[27] Nevertheless most groups now perform bilateral nephrectomy to remove nephritic kidneys.

The work of Kolff and associates has placed pre-transplant nephrectomy for cure of hypertension on a firm foundation. The resulting anephric state is easily managed by hemodialysis and the relief of previously uncontrollable hypertension allows less complex care of the patient.[12]

As with all surgery, single stage operations are preferable. Therefore, whenever possible a combination of recipient nephrectomy and renal transplant is done as a single procedure. Occasionally, immediate availability of a free kidney or sudden harvesting of a cadaver kidney provides a useful graft for a relatively unprepared patient. In this circumstance,

especially when accompanying factors cast doubt upon viability or function of the graft, the recipient nephrectomy can be deferred. The advantage gained is insurance of excellent renal function at the time of the bilateral nephrectomy.

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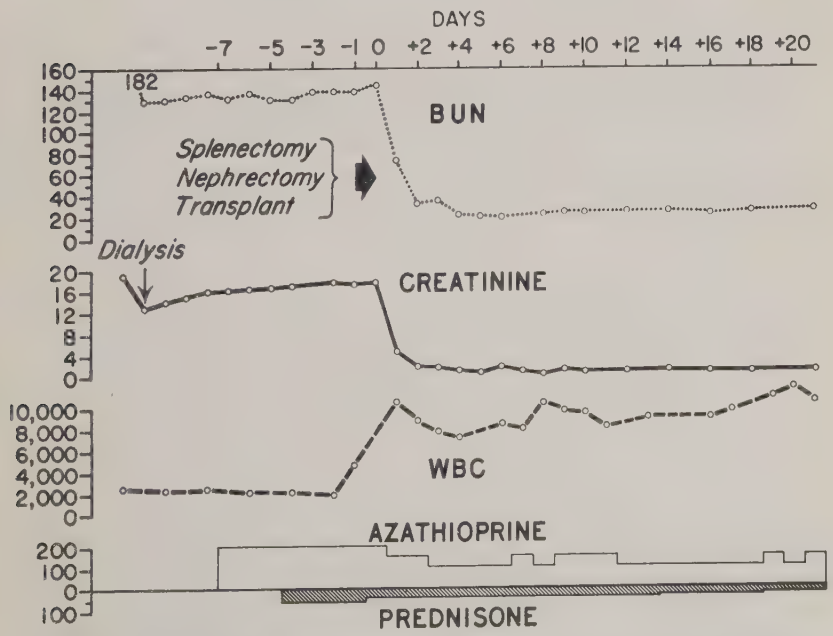

Figure 4. The pre-transplant preparation and early post-transplant progress is shown on this composite chart.

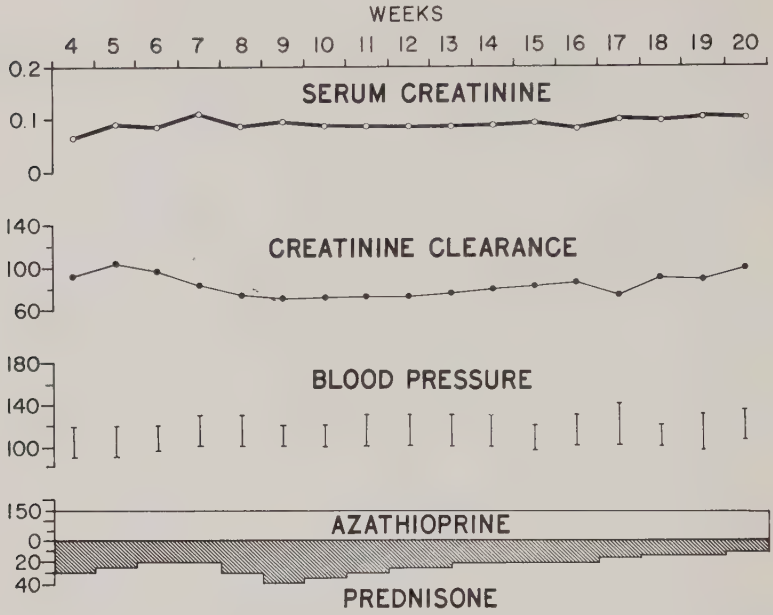

Figure 5. Stability of various physiologic functions of the graft are illustrated by this chart which depicts the first 20 post-transplant weeks of an 18 year old boy who received his mother's kidney. He is well 18 months postoperatively.

small lymphocytes which initiate immunologic reactions.[11] On theoretical grounds, thymectomy would seem to be an important part of renal allografting. The Denver group is currently attempting to assess this factor. Other transplantation teams are not as yet performing the procedure routinely.

Immune Suppression

Immune suppressive protocols are used with varying degrees of success by various transplant groups. Ignorance of methods of invocation of the immune response makes such protocols empiric (Fig. 4). Basic to current immunosuppression is the use of azathioprine (Imuran). This antimetabolite, an imidazole derivative of 6-mercaptopurine, acts to inhibit steps in purine synthesis, the conversion of phosphoribosylpyrophosphate to phosphoribosylamine and the synthesis of adenosine monophosphate. In this way it interferes with cell proliferation and presumably chiefly affects those cells concerned with initiation of the immune response. This explains its lack of generalized destruction of lymphoid tissue. Azathioprine is given in a dose range of 3 to 5 mg./kg./day (Fig. 5).

The corticosteroids exhibit a variety of effects including an interference with antigen uptake, phagocyte function, and delay in establishment of lymphatic connection between graft and host.[3] Their utility in modifying and even reversing established rejection reactions has been one of the encouraging features of clinical transplantation.

The actinomycins are also used to reverse rejection crises. These are peptide-containing antibiotics, the most useful of which, Actinomycin C, is a combination of three compounds. It is used in small doses for short periods of time and is known to interfere with messenger RNA synthesis by means of selectively inhibiting DNA-dependent RNA synthesis.

RESULTS

Combined reporting of results of kidney transplantation has been possible because of Murray's establishment of the Human Kidney Transplant registry. The fourth report of this registry has appeared in detail.[17] The data from it indicates that genetically related donors represent the best source of renal tissue from a functional if not a moral standpoint. For example, 72 per cent of identical twin transplants have survived one year (32 attempts). Similarly, 51 per cent of sibling grafts (72 attempts) and 38 per cent of other blood relative grafts (112 attempts) have lasted 12 months. However, Shackman's case of slow rejection at 45 months characterized by a vascular proliferative lesion in a sibling transplant is discouraging. Late failure in maternal, cadaver and dizygotic twin grafts as well as other factors is causing many groups to look with disfavor upon the utilization of living volunteers, sibling, blood relative or otherwise, as donors of kidneys.

As technical problems of procurement are solved, initial technical success has been achieved in a greater percentage of cadaver grafts. During the past two reporting periods which extend over 12 months, survival of one-half of the 123 attempted cadaver grafts has been accomplished. The ultimate fate of these grafts is unsettled but the previous overall one year survival of unrelated donor grafts and cadaver grafts is 10 per cent.

It has been established without doubt that cancer can be transplanted with functioning renal tissue. This, of course, was a questionable factor prior to its documentation by several transplant groups. Because of this, the use of cadaver organs from patients dying of disseminated malignancy at this time would be unwise. However, in Zukoski's review of 50 consecutive autopsies, at a Veterans Administration Hospital, 17 were found to have had the potential for yielding acceptable kidneys for transplantation.[6] While this result is a higher estimate than has been

available previously, it is not unrealistic. In fact, in pediatric or general hospitals, a higher figure might be expected.

COMMENT

There is a growing concern in medicine and among nonmedical observers of science regarding human experimentation. Since human kidney transplantation is experimental, the transplant teams engaged in this investigation must be aware of their role in the development of experimental ethic. The *Wall Street Journal* has indicated that "A controversy with highly significant overtones is smoldering among medical scientists over whether medical experiments involving human patients are in danger of exceeding ethical bounds." This concern is tempered by knowledge that "human experimentation now holds more promise of benefiting mankind than at any other time in history."

Perhaps most transplant teams would agree with Francis Moore, Mosely Professor of Surgery at Harvard, who states that "good science is ethical science." He gives some guidelines for use in application of new therapy indicating that the patient and his family must be educated to the various alternatives, that the patient must be given the best medical care available, that the doctors concerned have done preliminary laboratory work which has demonstrated their competence and skill, and that each patient's case be studied and documented as carefully as possible. Certainly, if these guidelines are followed in renal transplantation, this aspect of medical science will continue to progress toward a time when predictable clinical success will be a reality and transplantation can become therapeutic rather than investigational.

ACKNOWLEDGMENT

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REFERENCES

1. Aisenberg, A. C.: Drugs used for the suppression of immunologic responsiveness. New England J. Med. *272:* 114, 1965.
2. Anthony, C. W. and MacIntyre, I. H.: Laboratory costs of organ transplantation. J.A.M.A. *192:* 171, 1965.
3. Berenbaum, M. C.: Immunosuppressive drugs. Brit. M.J. *21:* 140, 1965.
4. Calne, R. Y.: Renal Trnnsplantation. Baltimore, Williams & Wilkins Co., 1963.

5. Couch, N. D., Curran, W. J., Hyg, S. M. and Moore, F. D.: The use of cadaver tissues in transplantation. New England J. Med. *271:* 691, 1964.
6. Ende, N. and Zukoski, C. F.: Emergency autopsy for the selection of donor kidneys. J.A.M.A. *191:* 902, 1965.
7. Haber, M. H. and Putong, P. B.: Ureteral vascular rejection in human renal transplants. J.A.M.A. *192:* 417, 1965.
8. Hamburger, J.: Experience with 45 renal homotransplantations in man. Lancet *1:* 985, 1965.
9. Hume, D. M., Merrill, J. P., Miller, B. F. and Thorn, G. W.: Experience with renal homotransplantation in the human: Report of 9 cases. J. Clin. Invest. *34:* 327, 1955.
10. J. R. E. (editorial): Moral problems in the use of borrowed organs, artificial and transplanted. Ann. Int. Med. *60:* 309, 1964.
11. Karetzky, M. and Rudolph, L. E.: Current status of the thymus gland. Surg. Gynec. & Obst. *119:* 1, 1964.
12. Kolff, W. J., Nakamoto, S., Poutusse, E. F., Straffon, R. A. and Figueroa, J. E.: Effect of bilateral nephrectomy and kidney transplantation on hypertension in man. Circulation *30:* 23, 1964.
13. Lawrence, W., Miller, D. G., Isaacs, M. and Whitmore, W.: Criteria for donor selection in human kidney transplantation. Transplantation *3:* 452, 1965.
14. McIntosh, D. A., McPhaul, J. J., Peterson, E. W., Harvin, J. S., Smith, J. R., Cook, F. E. and Humphreys, J. W.: Homotransplantation of a cadaver neoplasm and a renal homograft. J.A.M.A. *192:* 1171, 1965.
15. Marchioro, T. L., Brittain, R. S., Hermann, G., Holmes, J., Waddell, W. R. and Starzl, T. E.: Use of living donors for renal homotransplantation. Arch. Surg. *88:* 711, 1964.
16. Mims, M. M.: Hemodynamics of renal transplantation. J. Urol. *86:* 493, 1961.
17. Murray, J. E., Gleason, R. and Bartholomay, A.: Fourth Report of the Human Kidney Transplant Registry. Transplantation *3:* 689, 1965.
18. Murray, J. E. and Harrison, J. H.: Surgical management of fifty patients with kidney transplants including eighteen pairs of twins. Am. J. Surg. *105:* 205, 1963.
19. Murray, J. E., Merrill, J. P. and Harrison, J. H.: Kidney transplantation between seven pairs of identical twins. Ann. Surg. *148:* 343, 1958.
20. Rifkind, D., Marchioro, T. L., Waddell, W. R. and Starzl, T. E.: Infectious diseases associated with renal homotransplantation. J.A.M.A. *189:* 397, 1964.
21. Rifkind, D., Starzl, T. E., Marchioro, T. L., Waddell, W. R., Rowlands, D. T. and Hill, R. B.: Transplantation pneumonia. J.A.M.A. *189:* 808, 1964.
22. Starzl, T. E.: Experience in Renal Transplantation. Philadelphia, W. B. Saunders Co., 1964.
23. Starzl, T. E., Marchioro, T. L., Holmes, J. H., Hermann, G., Brittain, R. S., Stonington, O. H., Talmage, D. W. and Waddell, W. R.: Renal homografts in patients with major donor-recipient blood group incompatibilities. Surgery *55:* 195, 1964.
24. Starzl, T. E., Marchioro, T. L., Morgan, W. W. and Waddell, W. R.: A technique for use of adult renal homografts in children. Surg. Gynec. & Obst. *119:* 106, 1964.
25. Veith, F. J., Luck, R. J. and Murray, J. E.: Effects of splenectomy on immunosuppressive regimens in dog and man. Surg. Gynec. & Obst. *121:* 299, 1965.
26. Waller, M., Irby, R., Mullinax, F. and Toone, E. C.: Connective-tissue disease and rheumatoid factors in patients with renal transplants. New England J. Med. *273:* 12, 1965.
27. Woodruff, M. F. A., Robson, J. S., Nolan, B., Lambie, A. T., Wilson, T. I. and Clark, J. G.: Homotransplantation of kidney in patients treated by preoperative local irradiation and postoperative Imuran. Lancet *2:* 675, 1963.

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Pulmonary Embolectomy

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Pulmonary embolectomy epitomizes the idealistic goals of the surgeon in a most dramatic fashion. The surgeon, possessing incisive diagnostic acumen and precise prognostic judgment, can with swift and skillful action save a patient from certain sudden death and return him to his normal state of health. It is no wonder that the operation has captured the imagination of surgeons in spite of the fact that very few operations have been performed successfully.

Pulmonary embolectomy was first proposed by Trendelenburg in 1908.[9] He demonstrated its successful application in the experimental animal, but of his three patients operated, one died on the operating table, another died 15 hours postoperatively of heart failure and the last died 37 hours postoperatively of hemorrhage from the internal mammary artery. It remained for Kirschner, a pupil of his, to perform in 1924 the first clearly successful pulmonary embolectomy.[4] Throughout the world subsequent successful operations prior to this decade were rare; the first reported in this country was not until 1958.[7]

In the last five years, however, many more successful pulmonary embolectomies have been performed at many centers. This recent success can be due to an increased awareness of the disease and treatment, wide-

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spread application of resuscitative measures, development of more definitive diagnostic methods, but perhaps the most important factor has been the use of cardiopulmonary bypass. The first successful pulmonary embolectomy utilizing cardiopulmonary bypass was apparently performed by Sharp in 1961.[6]

DIAGNOSIS

The first problem for the surgeon is to make the diagnosis rapidly and with certainty. Submitting a patient with acute coronary occlusion to this operation is not only futile but very likely fatal to one who otherwise might survive with judicious supportive care.

The clinical diagnosis of massive pulmonary embolism is not difficult in the postoperative patient with antecedent thrombophlebitis who then suddenly complains of chest pain and rapidly develops dyspnea, apprehension, tachycardia, hypotension, syncope and cyanosis. However, most patients do not exhibit this classical picture. Most of the patients with pulmonary embolism are not even on the surgical wards. Our two youngest patients with massive pulmonary embolism were 26 and 29 years old. Most of the patients do not have signs of phlebitis. In general, the signs and symptoms depend upon the number and size of emboli, the rate of embolization and the prior state of health. These many variables produce a wide spectrum of clinical presentation, ranging from vague chest pain to sudden death.

If pulmonary infarction accompanies or precedes the massive embolization, pleuritic pain, fever, hemopytsis, friction rub, blood-tinged pleural effusion and jaundice may be present.

In acute massive pulmonary embolism the chest roentgenogram tends to be normal with subtle changes in the diameter of the pulmonary arteries or unilateral elevation of the diaphragm; the electrocardiogram tends to be normal but often shows nonspecific changes or occasionally right heart strain patterns; arterial oxygen desaturation may be somewhat resistant to oxygen administration; the arterial pCO_2 is significantly higher than the alveolar (or end tidal) pCO_2; and the macro-aggregated, radioiodinated albumin pulmonary scanning reveals large areas of absent or low blood flow.

Pulmonary arteriography is the only method, however, which has afforded us certain diagnosis in the shortest possible time. In addition, the severity of the pulmonary arterial obstruction is most accurately assessed by this method, which is performed as follows:

Under local anesthesia a peripheral vein is isolated and catheterized. The tip of the catheter is advanced to the main pulmonary artery and

a pressure is assessed. Then, with the catheter tip in the main pulmonary artery, a pulmonary arteriogram is taken (Figs. 1, *A*, 2, *A*). Massive pulmonary embolism is readily diagnosed and the degree of obstruction can be accurately assessed from the arteriogram and the pressure meas-

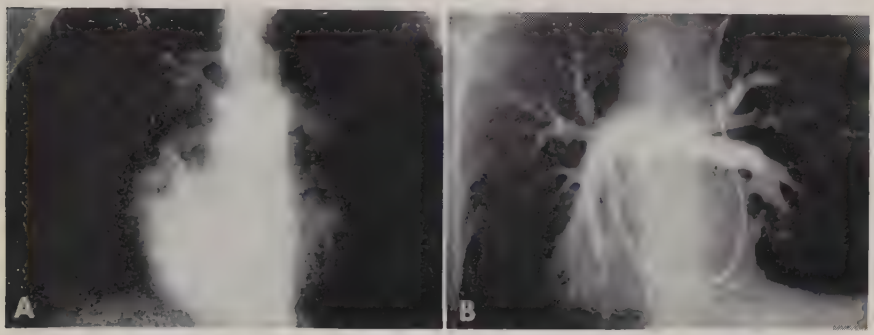

Figure 1. S.H., aged 26 years. *A*, This is one of many similar frames in an angiocardiogram showing stasis of radiopaque dye in the right ventricle and pulmonary artery with sparse filling of the left upper lobe arteries performed immediately prior to embolectomy.

B, Nearly normal pulmonary arteriogram performed 3 weeks after embolectomy.

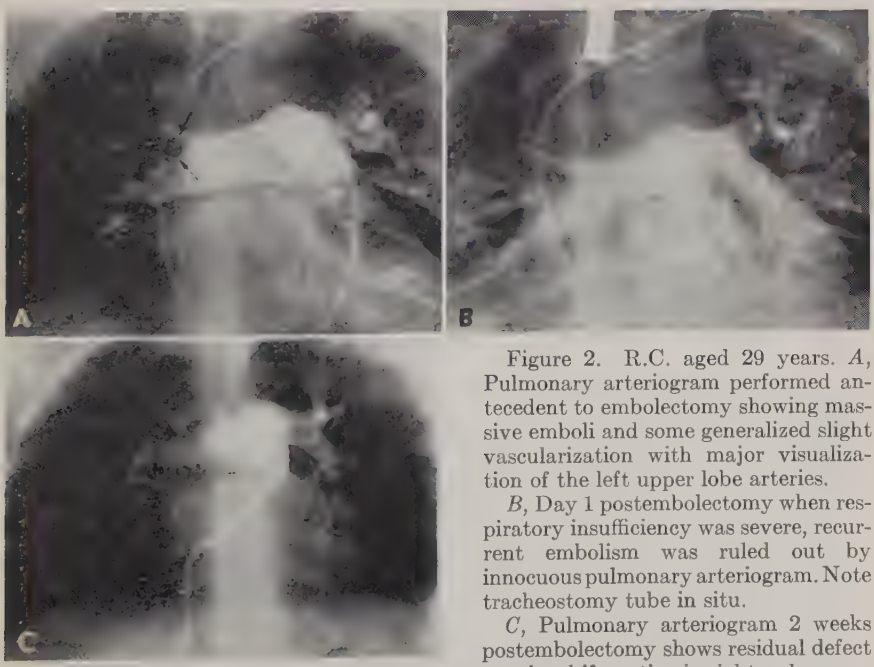

Figure 2. R.C. aged 29 years. *A*, Pulmonary arteriogram performed antecedent to embolectomy showing massive emboli and some generalized slight vascularization with major visualization of the left upper lobe arteries.

B, Day 1 postembolectomy when respiratory insufficiency was severe, recurrent embolism was ruled out by innocuous pulmonary arteriogram. Note tracheostomy tube in situ.

C, Pulmonary arteriogram 2 weeks postembolectomy shows residual defect at prime bifurcation in right pulmonary artery.

urement. A number of desperately ill patients, including some undergoing resuscitation, have been studied in this manner without fatality or morbidity attributable to the procedure.

INDICATIONS

Pulmonary embolectomy is reserved for the patient who will die without it. At the present time we do not believe that embolectomy would be justified on the basis of the chronic effects of massive embolization, but rather only on the immediate effects. Although evidence suggests that minute, repeated pulmonary emboli can result in chronic pulmonary hypertension, documentation of acute, massive pulmonary embolism causing persistent significant pulmonary hypertension is lacking. A few cases can apparently survive a massive embolism and be left with chronic, partially occluding, organized, thromboembolic masses which are productive of symptoms. On the other hand, experimental evidence, case reports[2] and personal experience have indicated that acute pulmonary emboli can be nearly completely lysed in a short time and leave no detectable abnormalities. This is probably the more common sequence and it has been shown to be significantly enhanced by appropriate anticoagulant therapy.

How then is the surgeon to decide which patient will die without embolectomy and which will survive and completely lyse the emboli? This is no doubt the most difficult question in the management of massive pulmonary embolism. The need for embolectomy seems quite clear in the patient who is unable to maintain an adequate cardiac output and blood pressure, but even here it is possible that the major embolus could impact a few millimeters deeper and open up an orifice of a lobar artery, altering the clinical picture dramatically. Conversely, another patient may have collapsed, rapidly recovered and when seen only demonstrate mild dyspnea, slight cyanosis, tachycardia and modest hypotension or vague chest discomfort. He may improve in a few hours, then suddenly collapse again and expire. Because of these difficulties it seemed prudent to reserve the operation for only those patients who had seemingly compensated fully to the emboli already lodged in their pulmonary arteries when first seen.

Experimental data indicate that up to about 75 per cent occlusion of pulmonary arterial system is tolerated; however, accurate assessment of the degree of occlusion is difficult when the occlusions are scattered widely and involve many various sized arteries. Arteriography and lung scanning both can give a rough approximation of the degree of occlusion.

Hemodynamic data from our laboratory have indicated that with

repeated emboli in dogs, full compensation and the prelethal state is predicted best by the increase in pulmonary vascular resistance and next by the mean pulmonary artery pressure to mean systemic artery pressure ratio.[3] In a somewhat steady state, when the mean pulmonary arterial/systemic arterial pressure ratio was 26 per cent ± 5 per cent, the dog could accept another 10 cc. embolus with survival, but when the ratio was 39 per cent ± 9 per cent, the next embolus was fatal. Other hemodynamic parameters were not statistically significant, including changes in total pulmonary blood volume. Since the pressure ratio can be quite readily obtained at the time of pulmonary arteriography, it has been used in preference to the pulmonary vascular resistance calculation which requires a cardiac output determination. Therefore, fairly accurate assessment of the degree of obstruction and compensation can be obtained rapidly by pulmonary arteriography and blood pressure measurements, and embolectomy can be reserved for the more desperate and potentially fatal cases.

At the present time our indications for pulmonary embolectomy are as follows:

1. Clinical indications of severe pulmonary arterial obstruction (e.g. tachycardia, hypotension, dyspnea, cyanosis, chest pain, collapse, etc.)
2. Acute episode of one week or less.
3. Nearly 75 per cent obstruction of the main and lobar pulmonary arteries by arteriography.
4. Mean pulmonary arterial pressure greater than 30 per cent of mean systemic arterial pressure.

TECHNIQUE (Fig. 3)

The easiest job for the surgeon is the performance of the pulmonary embolectomy utilizing cardiopulmonary bypass. Since time is important, it is essential to anticipate this problem and have the appropriate sterilized oxygenator and equipment available. The disposable bubble oxygenators are ideal for this purpose. Following arteriography, if the clinical signs and pressures are indicative of massive embolism, the patient is transferred to the operating room and blood is cross-matched while the pulmonary arteriograms are being processed. The arteriographic catheter can be withdrawn so that its tip will be in the superior vena cava for the administration of intravenous agents and subsequent central venous pressure monitoring.

Although we are quite prepared to place the patient on partial bypass via peripheral cannulations, we have been able to bring the patient to the operating room and begin total bypass in the standard manner

without apparent deterioration of the patient's condition. Furthermore, it has been considered inadvisable to cannulate a potentially diseased or clot-containing femoral-iliac venous unit. These patients have usually had a wide variety of medication given initially and at this point probably require only 100 per cent oxygen and a vasopressor. (There is experimental but not clinical evidence that isoproterenol is preferable to the usual vasopressors.) The diagnosis is established with arteriography by this time (Figs. 1, *A*, 2, *A*), and the need for embolectomy defined.

Minimal amounts of anesthetic agents are necessary and the trachea is easily intubated. Simultaneous groin and median sternotomy incisions are performed. The patient is given heparin (300 units/kilogram body weight) intravenously. A large catheter is quickly inserted in the right atrium and with the femoral artery cannulated partial bypass is initiated. The patient's color improves, mean arterial pressure rises, the distended right heart empties and more anesthetic agent becomes necessary as the state of consciousness rapidly rises. Completion of caval cannulations with institution of complete bypass follows and preparation of pericardial suspension and opening of both pleural cavities are performed during a stabilization and myocardial resuscitation period. It is altogether possible to perform this operation using dextrose solution priming of the pump oxygenator; however, significant blood replacement is usually necessary. The incisions, having been made rapidly in a hypotensive, vasoconstricted patient who is now heparinized, begin to bleed freely. It is helpful to have cardiotomy suction available to preserve the patient's own blood.

The main pulmonary artery is opened longitudinally and the emboli are removed first from the main, left and right pulmonary arteries with the delicate use of right-angled forceps, sponge forceps, uterine forceps or gallstone forceps. Every effort should be made to remove these clots intact so as to include their long peripheral tails. Passing a suction catheter down both pulmonary arteries is also a useful maneuver. This is combined with irrigation. A third and important maneuver was first suggested by Cooley, Beall and Alexander.[1] Both pleural spaces are opened and the lungs manually massaged. This combined with extraction maneuvers produces a surprising amount of additional clots. Retrograde flushing via the pulmonary veins has been recommended, but we have had no experience with this method. Before closing the pulmonary artery the right ventricular cavity is examined and suction is applied as a check for emboli lodged in chordae tendinae. The inferior caval tape is loosened and the abdomen compressed to retrieve any late, loose clots. The cannulae are removed and the patient is given protamine.

As hemostasis of the primary wound is being secured, a right lateral abdominal incision is made. Through an extraperitoneal plane the inferior vena cava is isolated and ligated infrarenally with a dacron tape.

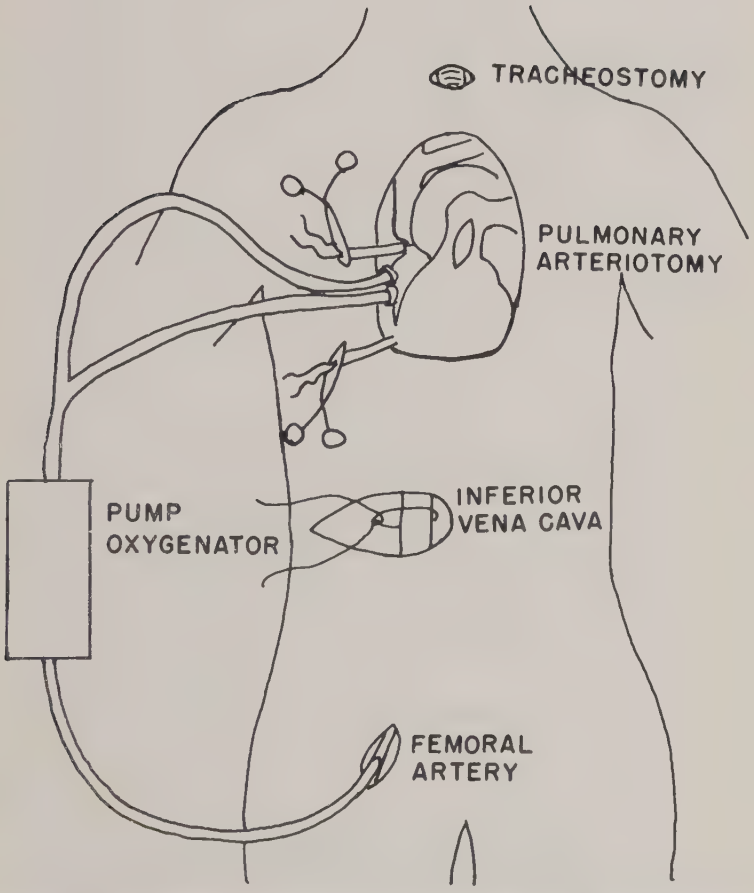

Figure 3. This drawing is a composite of the operations, indicating the method of cardiopulmonary bypass, longitudinal pulmonary arteriotomy, site of the inferior vena caval ligation and the tracheostomy.

The perfusion catheters are removed, mediastinal and multiple bilateral pleural drains are placed, the wounds are closed and a tracheostomy is performed for insertion of a cuffed tube (Fig. 3).

POSTOPERATIVE CARE

The final phase of the problem can be the most demanding of the surgeon's skill and attention. The postoperative care of these patients is as difficult as that of any desperately ill cardiac patient postperfusion.

Most of the patients requiring embolectomy will have had a preoperative period of severe tissue ischemia due to low systemic pressure, high venous pressure and low systemic blood flow. The metabolic acidosis usually will require correction with sodium bicarbonate or trishydroxyaminomethane.[5, 8] Other consequences of this ischemia may appear in the postoperative period and require specific attention (e.g., cerebral edema, renal failure, hepatic insufficiency, gastrointestinal bleeding, myocardial infarction, etc.).

Although the patient seemingly may have had a complete pulmonary embolectomy as judged by immediate postoperative arteriography (Fig. 2, *B*) and a normal pulmonary artery pressure, there remains evidence of considerable respiratory insufficiency. In spite of administering oxygen in high concentrations (pO_2 of 300 to 400 mm. Hg), the arterial pO_2 falls below 100 mm. Hg when there is sufficient ventilation to obtain a near normal pCO_2. This respiratory problem can have the features of veno-arterial shunting, faulty diffusion and hypoventilation.

In any one patient the respiratory insufficiency may be due to any single factor or combination of these factors. Fortunately, the administration of high concentrations of oxygen with a respirator improves the oxygenation of arterial blood in this situation. Occasionally, hyperventilation becomes necessary as can be seen in one of our postoperative patients (Fig. 4). During the fourth and fifth determinations of blood gases when the respirator was first used, the pCO_2 was near 20 mm. Hg, with normal buffer-base balance indicating hyperventilation. Yet, the inspired pO_2 was approximately 250 mm. Hg greater than the arterial pO_2. This respiratory insufficiency may last up to a week and for this reason a tracheostomy tube is a necessity.

Lastly, standard measures for the prevention of lower extremity thrombophlebitis, whether or not it was evident preoperatively, include continuous mild elevation of the feet until the patient is ambulatory, elastic supports to the thighs when he is ambulatory, and warfarin for at least 6 to 12 months. Well after recovery a right heart catheterization is performed and pulmonary arteriogram made to assess the extent of

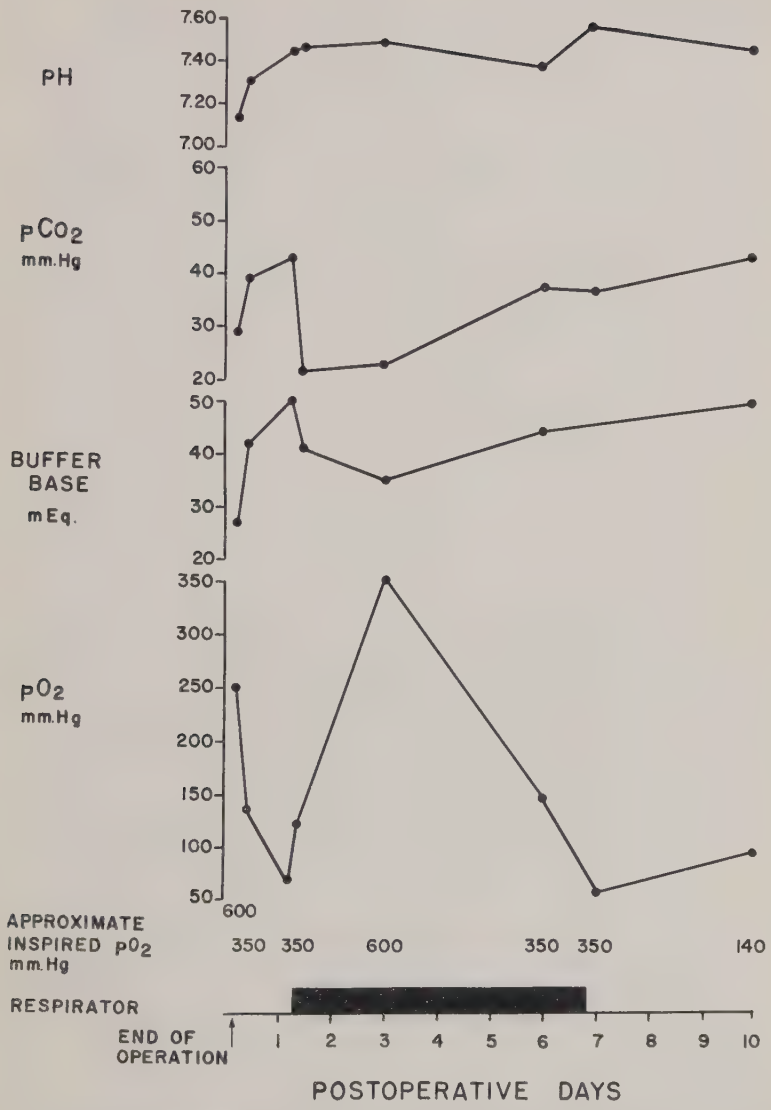

Figure 4. This graph indicates the arterial blood gas studies in a patient who had undergone pulmonary embolectomy. The initial metabolic acidosis was easily corrected with Tham. Not included in the graph was an attempt to take the patient off the respirator on the sixth day. The pCO_2 rose to 80.6 mm. Hg and the pO_2 fell to 38.9 mm.Hg in spite of the administration of 50 per cent (350 mm.Hg) oxygen with a fitted prosthesis over the tracheostomy tube.

return to normal adjustment of personal work and other activities (Fig. 1, *B*, 2, *C*).

SUMMARY

1. Massive pulmonary embolism produces a wide clinical spectrum.

2. Absolute diagnosis can be made safely by immediate pulmonary arteriography.

3. The need for pulmonary embolectomy is probably best predicted by the degree of occlusion as seen on the arteriogram and the pulmonary/-systemic arterial blood pressure ratio.

4. The operation can be performed rapidly with predictable survival utilizing cardiopulmonary bypass.

5. Adequate postoperative oxygenation may require a long period of assisted ventilation with high concentrations of oxygen.

REFERENCES

1. Cooley, D. A., Beall, A. C. and Alexander, J. K.: Acute massive pulmonary embolism. J.A.M.A. *177:* 283, 1961.
2. Freiman, D. G.: Pathologic observations on experimental and human thromboembolism in Pulmonary Embolic Disease (A. A. Sasahara and M. Stein, Eds.). New York, Grune & Stratton, 1965, pp. 81–85.
3. Greenburg, A. G., Daicoff, G. R., Toporoff, S. J. and Moulder, P. V.: The lethal pulmonary embolus. S. Forum, 1965.
4. Kirschner, M.: Ein durch die Trendelenburgsche Operation geheilter Fall von Embolie der Art. pulmonalis. Arch. klin. Chir. *133:* 312, 1924.
5. Moulder, P. V.: Pulmonary hypertension. Postgrad. Med. *37:* 686, 1965.
6. Sharp, E. H.: Pulmonary embolectomy: Successful removal of a massive pulmonary embolus with support of cardiopulmonary bypass—case report. Ann. Surg. *156:* 1, 1962.
7. Steenburg, R. W., Warren, R., Wilson, R. E. and Rudolph, L. E.: A new look at pulmonary embolectomy. Surg. Gynec. & Obst. *107:* 214, 1958.
8. Thompson, R. G., Moulder, P. V., Harrison, R. W. and Daicoff, G. R.: Trometh-amine (Talatrol) for acidosis therapy in surgery. S. Clin. North America *43:* 179–186, 1963.
9. Trendelenburg, F.: Über die operative Behandlung der Embolie der Lungenarterie. Zentralbl. f. Chir. *35:* 1, 1908.

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The Technique of Emergency Portacaval Shunt

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The creation of a venous shunt between the portal and systemic circulation was first undertaken in 1877 in dogs by Nicholas Eck,[3] who suggested that it be tried in the human being for the relief of ascites. The operation was first accomplished in the United States in man by Whipple, Blakemore and Lord of New York for the treatment of portal hypertension. Their early reports were published in 1945.[1, 4]

A description of portacaval shunt as applied to the emergency treatment of patients bleeding from esophagogastric varices was selected to appear in this volume because of certain variations in the procedure designed to make the operation practical for poor-risk patients. The operative procedure to be described has two purposes; one is to achieve as large a shunt as possible and the second is to minimize trauma and blood loss by keeping the patient on the operating table for as short a time as possible. These factors and the patient's hepatic reserve are the principal considerations governing surgical mortality.

The authors have used emergency portacaval shunt for patients who bleed massively from esophagogastric varices and in whom adequate control cannot be obtained by nonoperative means. Sengstaken balloon tamponade often is effective for short periods in these patients, but rebleeding takes place when the balloons are deflated, and surgery becomes necessary as the last resort to prevent death from exsanguinating

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hemorrhage. Our experiences with transesophageal ligation of varices via a thoracic or abdominal approach, and with high gastric ligation of varices through a gastrotomy incision have led us to abandon these procedures in favor of the emergency portacaval shunt.

The technique to be described concerns adult patients with alcoholic cirrhosis.

DIAGNOSTIC CONSIDERATIONS

Though bleeding from esophagogastric varices may be uncontrollable, it usually is a persistent ooze, less often a brisk hemorrhage, only rarely a massive exsanguination. Blood replacement can usually be maintained at a sufficient rate to prevent shock while diagnostic procedures are undertaken.

The diagnosis depends upon a history of alcoholism and the stigmata of Laennec's cirrhosis. Confirmatory tests are the 45-minute bromsulphalein test and upper gastrointestinal x-ray series after barium swallow. Esophagoscopy should be done when the patient's condition permits.

Splenoportography is helpful in determining whether there is a portal vein suitable for anastomosis. It carries a risk of hemorrhage from the spleen even in the most experienced hands. The risk is increased when one is dealing with a restless, actively bleeding patient who is in a state of ammonia intoxication and may be uncooperative. Our opinion that this procedure can be avoided is strengthened by the fact that in 84 consecutive patients with alcoholic cirrhosis upon whom we have performed portacaval shunt, we did not encounter a portal vein unsuitable for anastomosis, nor have we begun the operation and found that it could not be completed.

When Sengstaken balloon tamponade is ineffective in the control of hemorrhage, the physician should suspect that bleeding is not from varices but from a peptic ulcer or gastric erosion associated with a hiatus hernia. Here esophagoscopy and emergency upper gastrointestinal roentgenography may be particularly helpful. In some patients it may be impossible to identify with certainty the site of bleeding. No bleeding point may be seen on esophagoscopy, and there may be no evidence of peptic ulceration on roentgenography of the stomach and duodenum. Gastric varices may be the source of hemorrhage in some of these patients. In some of our patients the source of hemorrhage may have been a small gastric or duodenal ulcer which the roentgenologists were unable to visualize. It is our clinical impression that on occasion the bleeding from a peptic ulcer in patients with portal hypertension may be venous in

origin and may cease after adequate decompression of the portal system to normal or near normal levels of pressure.

PREOPERATIVE PREPARATION

The most important step in preoperative preparation is adequate blood replacement to prevent shock. These patients have an expanded blood volume often with a normal red cell mass and a low hematocrit. The determination of blood volume preoperatively provides an important baseline for pre- and postoperative replacement therapy. Blood volume should be restored using as a baseline the blood volume obtained for the patient who is being treated and not the normal for patients of the same weight. When many transfusions are given, every third or fourth one should contain fresh blood to supply prothrombin.

ANESTHESIA

Preoperative medication consists of 0.6 mg. atropine sulfate. No sedation is necessary in most of these patients because of ammonia intoxication. Short-acting barbiturates and meperidine (Demerol) are avoided preoperatively because these drugs are detoxified in the liver and have an enhanced effect when there is hepatic insufficiency.

General anesthesia is used. Induction is accomplished with cyclopropane and oxygen and minimal amounts of thiopental (Pentothal), 100 to 200 mg. Succinylcholine, 30 to 40 mg., is given to facilitate endotracheal intubation. Maintenance anesthesia is achieved with cyclopropane, oxygen and succinylcholine. It is important to maintain adequate concentrations of oxygen in the arterial circulation to prevent further liver damage. A mechanical respiratory assistor is used. Central venous pressure and electrocardiographic monitoring are maintained throughout the operative and postoperative period.

The anesthesiologist records the amount of blood lost as determined by the weighed sponge technique after all ascitic fluid has been aspirated.

We have had no experience with hypothermia or controlled hypotension.

OPERATIVE TECHNIQUE

The patient is positioned on the operating table in a true lateral position as shown in Figure 1 with the flank over the kidney rest, which

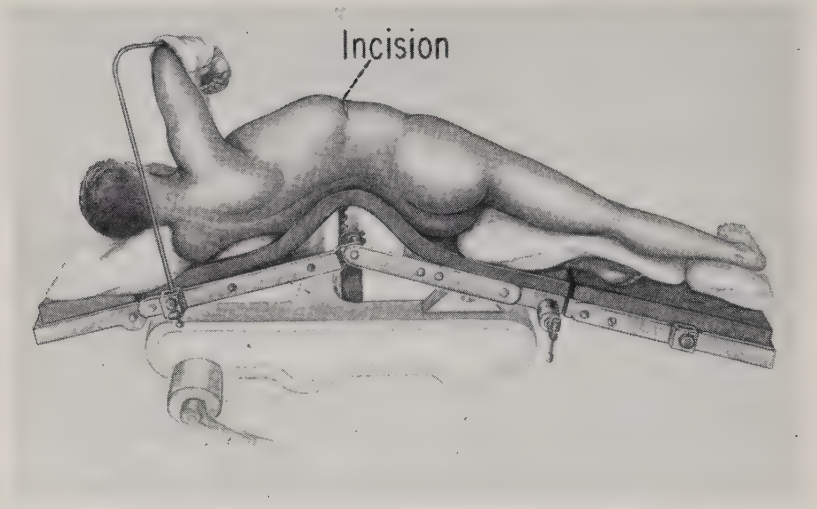

Figure 1. Position of patient on operating table for portacaval shunt.

will later be elevated. A jackknife position of the table is obtained after the incision is made to facilitate exposure. The lateral position causes the viscera in the upper abdomen to fall to the left, and this further aids in exposure of the operative area.

A subcostal incision is made with the skin incision carried above the costal border as shown by the straight line in Figure 2. The skin and subcutaneous tissues are then retracted caudally and the muscle, fascia and peritoneum are incised about 1 inch below the costal border. This incision resists disruption, particularly in patients who accumulate ascitic fluid postoperatively. If there is a leak of ascitic fluid, the leak seals within a few days. There have been no wound complications other than minor stitch infections in 75 patients in whom this incision was used.

The first maneuver on entering the abdomen is to identify the portal vein. This is done by inserting the index finger of the left hand into the foramen of Winslow and palpating the vein through the peritoneum. The compressibility of the vein with re-expansion when the finger is released enables the operator to identify it. The peritoneum on the superior margin of the foramen of Winslow is grasped with a long-handled Mixter forceps so that the peritoneum directly beneath the vein is brought into view by retracting the hepatoduodenal ligament medially (Fig. 3). The peritoneum is incised at this point and the incision lengthened in a cephalad direction. Later it is lengthened in the direction of the duodenum, taking care to avoid the mass of lymph nodes which lie adjacent to the portal vein at the point where the vein lies beneath the duodenum. These lymph nodes are retracted anteriorly and are avoided during the remainder of

the dissection. The finger is introduced into this incision to free the portal vein, which is then encircled with a rubber tubing (Fig. 4). Occasionally the coronary or the pyloric vein, which enter the portal vein along its medial aspect, will require ligation. If the incision in the peritoneum is carried too high the cystic vein may be cut and cause troublesome bleeding. These small tributaries of the portal vein should be individually ligated and divided. Occasionally they may be torn in the haste of an emergency dissection. If so, the operator should proceed with the exposure and then with the shunt, since decompression of the portal system is usually attended by cessation of bleeding from these vessels as soon as the pressure is lowered. The hepatic and cystic arteries and the bile ducts are not exposed or separately identified. They are retracted anteriorly with a Cushing retractor as shown in Figure 5.

A distended gallbladder may require decompression to facilitate exposure of the portal vein. Usually the gallbladder can be retracted behind a Dever retractor and does not require decompression.

The stomach and duodenum should be examined to determine if a peptic ulcer, hiatus hernia or other lesions are present, and the gallbladder should be palpated for stones. In the interest of completing the operation

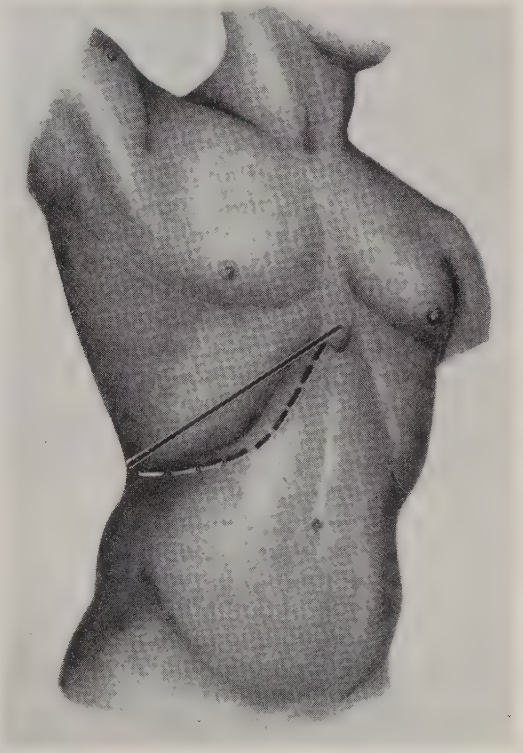

Figure 2. The solid line indicates the incision through the skin and subcutaneous tissue. The incision through the fascial and muscular layers is made beneath the costal border as indicated by the broken line.

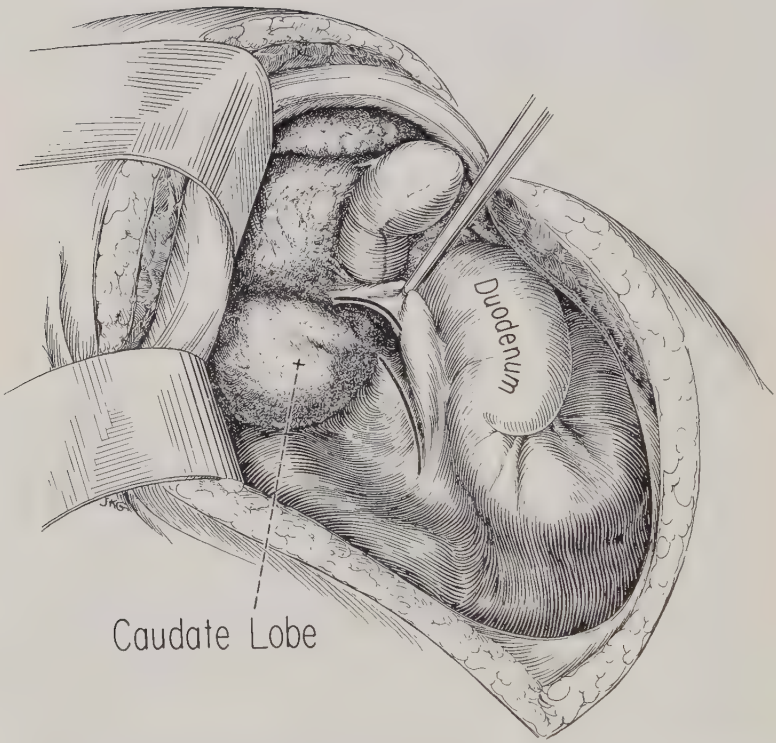

Figure 3. A solid line indicates the position of the incision through the peritoneum on the superior border of the foramen of Winslow to expose the portal vein. The lower line indicates the incision through the peritoneum to expose the vena cava. A caudate lobe may lie between the portal vein and the vena cava. Usually this lobe can be retracted upward to allow the shunt to be made beneath the lobe.

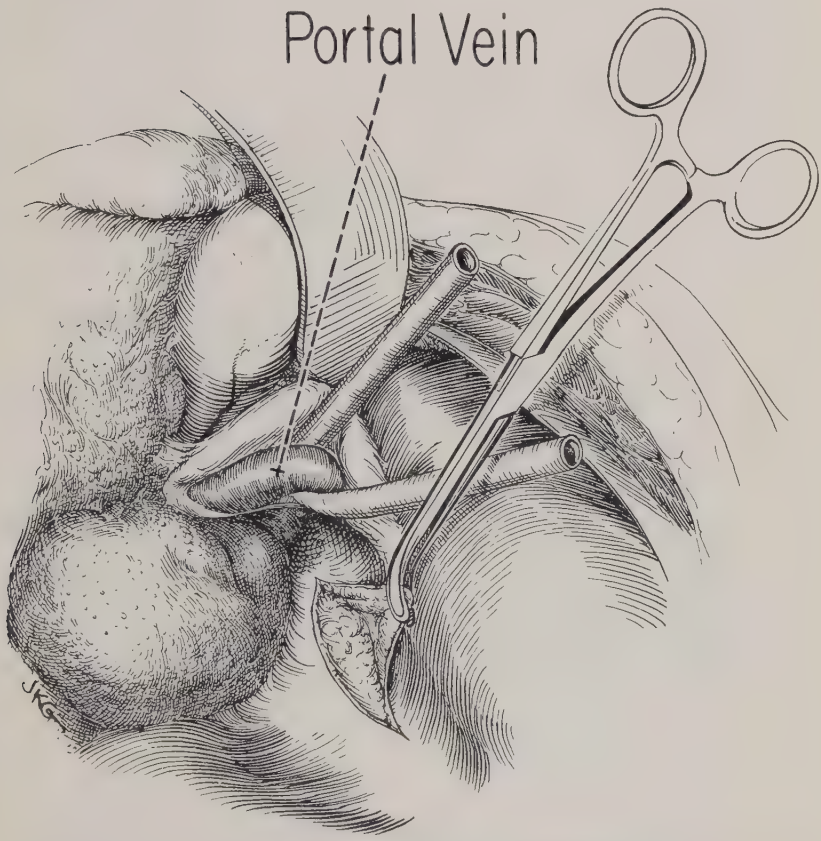

Figure 4. The portal vein is encircled with a rubber tubing. An incision through the peritoneum is then made to expose the vena cava.

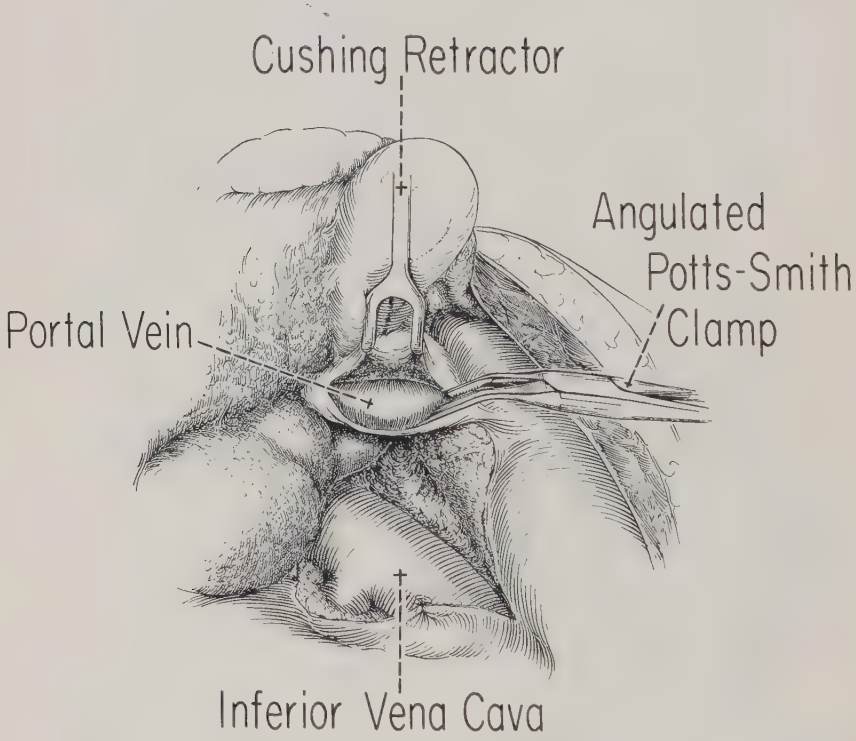

Figure 5. The portal vein is occluded with an angulated clamp. The portal vein is ligated and stick-tied as close to its entrance into the liver as possible.

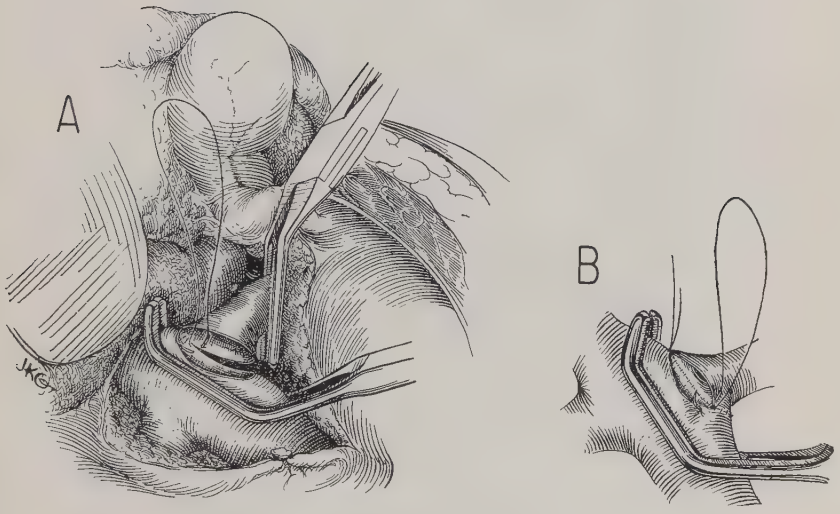

Figure 6. Construction of the anastomosis.

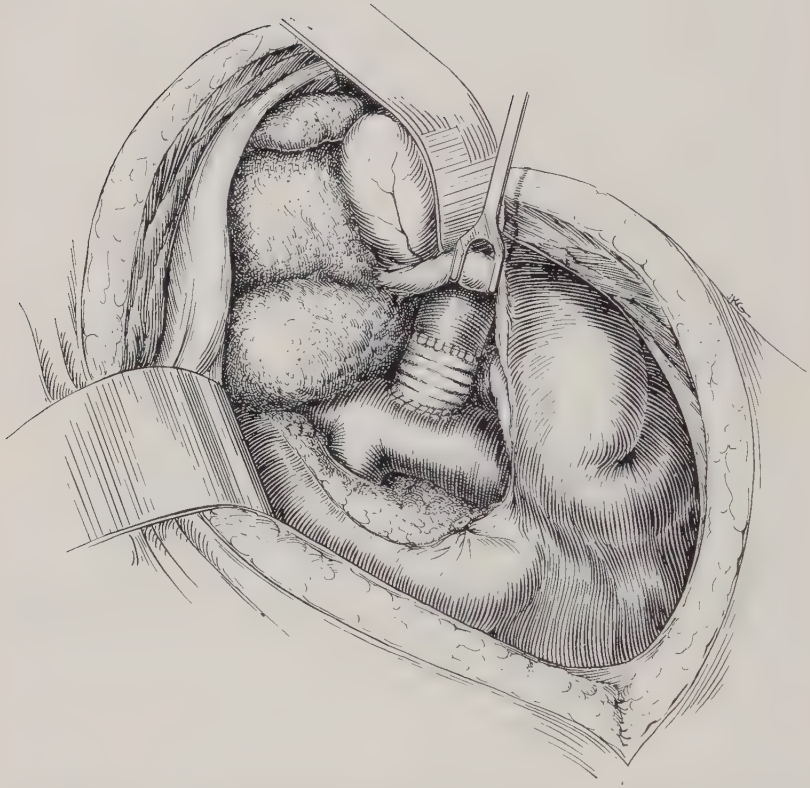

Figure 7. A Teflon prosthesis may be inserted between the portal vein and the vena cava if the portal vein is too short to reach the cava and to permit an anastomosis to be made without tension.

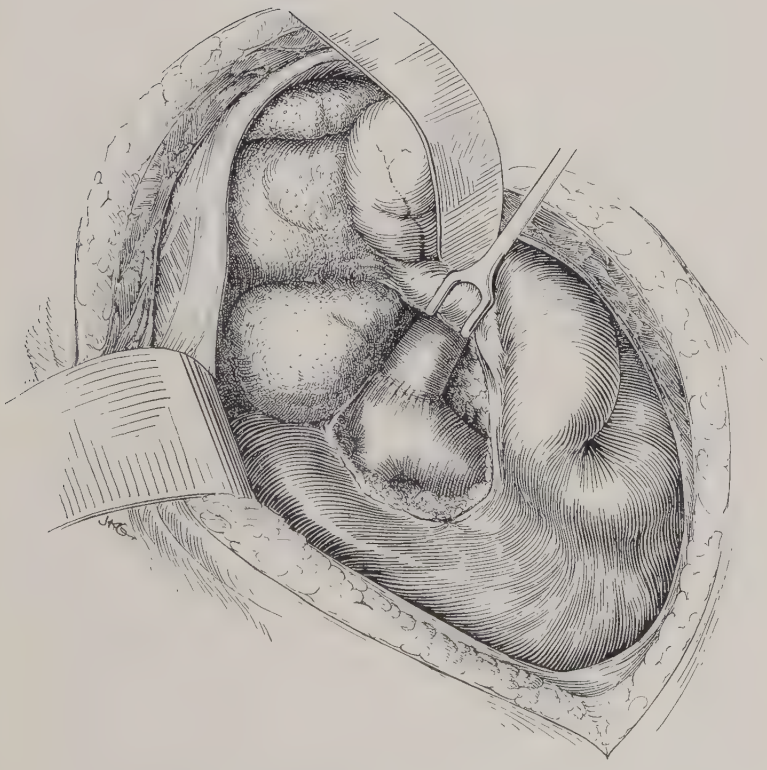

Figure 8. The completed portacaval shunt.

in as short a time as possible, we defer other operative procedures for lesions which may need surgical correction until the patient has recovered from the shunt.

After the portal vein has been mobilized from its point of emergence from beneath the pancreas to its division into left and right branches at its entrance into the liver, the vena cava is exposed by an incision through the peritoneum overlying it (Fig. 4). The superior surface of the vein is cleaned of connective tissue and fat, using sharp and blunt dissection. A forceps holding a cotton pellet is useful in this part of the dissection. The vena cava is exposed from a point about 1 cm. below the entrance of the right renal vein to the point of entrance of the lowest hepatic vein.

Pressure in the portal vein is taken by inserting a needle directly into the portal vein. A calibrated cerebrospinal fluid manometer containing dilute heparin in physiologic saline solution is connected to the needle and the pressure is read after the column of fluid has stabilized.

The portal vein is ligated with an 0 silk suture as close to the liver as possible. A 00 silk stick tie is placed proximal to the ligature for additional security, since dislodgement of the tie after the portal vein stump has retracted into the hilus of the liver may lead to profuse hemorrhage and the vein stump may be difficult to retrieve. A Potts-Smith angulated clamp or a Brock auricle clamp is then put on the portal vein (Fig. 5) and the vein divided in a right angle or slightly oblique direction, allowing at least 2 cm. of vein distal to the clamp but more if possible. A point on the anteromedial aspect of the vena cava is selected for the site of anastomosis and a grooved Bailey modified Satinsky clamp is placed on the cava to achieve partial occlusion (Fig. 6). A Satinsky clamp which does not have grooves may permit the vena cava to slip out of the clamp and is unsatisfactory. A button of vena cava is removed with a sharply curved scissors.

In some instances, the portal vein may not be sufficiently long to reach to the vena cava and to permit the construction of an anastomosis without tension. In these cases we are prepared to insert a short Teflon graft to bridge the gap (Fig. 7). This graft should be wider than it is long. We have done this in two elective portacaval shunts, one of which was open two years later at autopsy after the patient died of hepatic insufficiency. The other is presumably open in a patient two years postoperatively who is in satisfactory condition and has not bled since his shunt.

Occasionally a large caudate lobe lies between the portal vein and the vena cava. This can be reflected anteriorly and held by a retractor, allowing the anastomosis to be made behind the lobe. We have not found it necessary to wedge or to resect the lobe.

The anastomosis is made with a 5–0 Tevdek continuous suture (Fig.

6). After completion of the posterior suture line, it is important to release the occluding clamp on the portal vein temporarily to make sure that the portal vein is patent. Usually a brisk escape of blood occurs and the clamp is promptly retightened. If a brisk flow of blood does not occur, the vein should be explored. On two occasions we have removed clots from the vein and then achieved a satisfactory flow before the anastomosis was completed. After the final suture is placed, the clamp on the vena cava is released, then the clamp on the portal vein. A laryngeal mirror heated in warm water is used to inspect the posterior aspect of the anastomosis for bleeding points. The size of the anastomosis is measured with a steel caliper. Figure 8 shows the completed shunt.

Occasionally, to avoid kinking of the portal vein and to obtain extra length, the posterior fibrous connections of the pancreas can be divided. This is a vascular area and bleeding may be encountered, but it is usually low pressure portal bleeding which tends to stop after the portal system is decompressed.

The post-shunt portal pressure is determined by inserting the needle of the manometer into the portal vein or through the wall of the vena cava with manipulation of the point of the needle into the portal vein.

A Penrose drain is placed to the region of the shunt. A sump drain is placed beneath the right lobe of the liver and suction is applied for the removal of blood and ascitic fluid for the first 24 postoperative hours. Closure of the abdomen is done in layers with continuous catgut in the peritoneum and fascia reinforced with interrupted sutures of catgut or silk. Wire retention sutures are placed through all layers of the abdominal wall and skin, avoiding the ribs.

Of 31 patients subjected to emergency portasystemic shunt, the operation was completed in less than two hours in 6, in two to three hours in 14, and in more than three hours in 11.

POSTOPERATIVE CARE AND COMPLICATIONS

Postoperative Care

Oxygen is given by nasal catheter or with a respirator. Sedation and medication for pain should be avoided in order to promote adequate respiration and prevent aspiration of secretions. The barbiturates are particularly to be avoided since they are not readily detoxified in the presence of hepatic insufficiency.

Drains should be removed 24 hours postoperatively. Otherwise a tract may form through which ascitic fluid may be lost.

The patient may be out of bed when the pulse is below 90 per minute, usually on the second or third postoperative day.

Usually oral feeding can be started on the third postoperative day.

Thirty milliliters per hour of liquids (tea, bouillon, or water with sugar added) are given the first day of feeding; 60 ml. per hour the next day and 90 ml. per hour the following day. Full liquid diet may be given subsequently with progression to a diet containing 500 mg. of sodium chloride and 70 grams of protein per day. Vitamins B, C and K should be given for the first 30 days postoperatively. Mineral oil should be given by mouth (45 ml.) on the third postoperative day to promote bowel action.

Postoperative ileus may lead to ammonia production in the gut with rapid development of ammonia intoxication. Ileus may require nasogastric suction with a Levin or Miller-Abbott tube, but intubation may cause bleeding from esophageal varices and should be used only as a last resort.

Accurate recording of intake and output should be continued postoperatively for 30 days or until the patient goes home from the hospital.

Stitches should not be disturbed for at least 14 days. Retention sutures are left in place for 21 days. Wounds in these patients heal slowly and wound separation may occur.

Tracheal aspiration should be done carefully and with proper attention to routines. Frequent aspiration may be essential in the prevention of aspiration pneumonitis. Too frequent or rough aspiration or the use of too much suction may lead to damage of the laryngeal or tracheal mucous membrane with subsequent laryngitis, tracheitis and hemorrhage from these sites. Tracheostomy may be necessary in order to accomplish adequate endotracheal suction. However, we prefer to use it only as a last resort. Most of these patients are prothrombin deficient and tend to bleed. We have observed oozing from the edges of a tracheostomy incision which led to aspiration of blood into the lungs and pneumonitis from which the patient failed to recover.

Operative Mortality

Among 31 patients who underwent emergency portasystemic shunt, there were 30 portacaval shunts and one splenorenal shunt. The 30-day hospital mortality was 35.5 per cent. The commonest cause of death was hepatic failure often in association with continued or recurrent bleeding from esophageal or gastric varices (Table 1).

Table 1. *Principal Causes of Death Within 30 Days of Emergency Portasystemic Shunt*

Hepatic failure, continued bleeding from proven esophageal or gastric varices	4
Hepatic failure	4
Hepatic failure, continued bleeding presumably from esophageal varices	1
Continued bleeding from esophageal varices, bacteremia, pneumonitis	1
Shock, hypotension, anoxia, died at completion of operation	1

Table 2. *Thirty-Day Hospital Mortality for Emergency Portasystemic Shunt According to Hepatic Reserve*

GROUP	NUMBER OF PATIENTS	MORTALITY	
		Number	Per Cent
A. Minimal impairment.....	3	0	0
B. Moderate impairment.....	12	2	16.7
C. Advanced impairment.....	16	9	56.2
TOTAL.....	31	11	35.5

Nine out of eleven postoperative deaths were among 16 patients with advanced liver impairment. Table 2 presents the 30-day hospital mortality according to the classification of liver impairment as described by Child.[2]

Postoperative Bleeding

Hemorrhage during the postoperative period occurred most often in patients whose shunt measured 1.8 cm. or less in its greatest diameter, as shown in Figure 9. In six patients in whom the shunt measured 2.5 cm. or more there was only one mild postoperative hemorrhage, whereas hemorrhage occurred in six of seven patients whose shunt measured

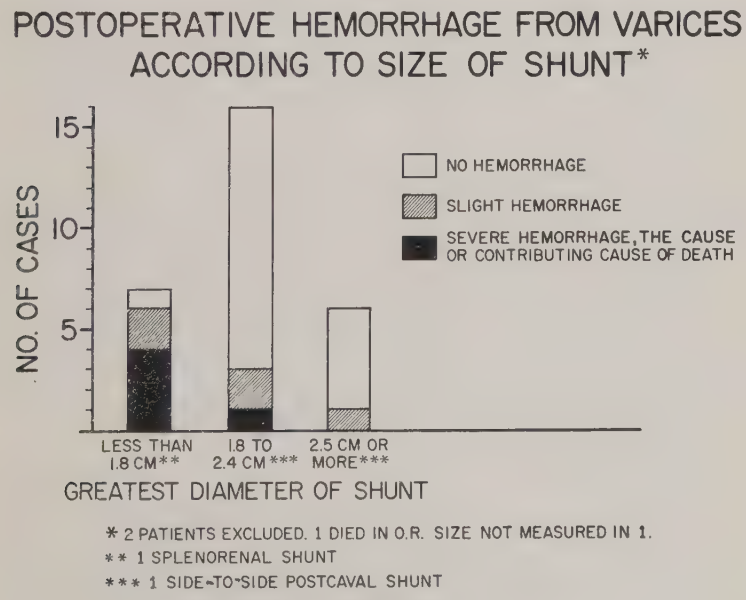
POSTOPERATIVE HEMORRHAGE FROM VARICES ACCORDING TO SIZE OF SHUNT*
NO. OF CASES
15
10
5
NO HEMORRHAGE
SLIGHT HEMORRHAGE
SEVERE HEMORRHAGE, THE CAUSE OR CONTRIBUTING CAUSE OF DEATH
LESS THAN 1.8 CM**
1.8 TO 2.4 CM***
2.5 CM OR MORE***
GREATEST DIAMETER OF SHUNT
* 2 PATIENTS EXCLUDED. 1 DIED IN O.R. SIZE NOT MEASURED IN 1.
** 1 SPLENORENAL SHUNT
*** 1 SIDE-TO-SIDE POSTCAVAL SHUNT

Figure 9

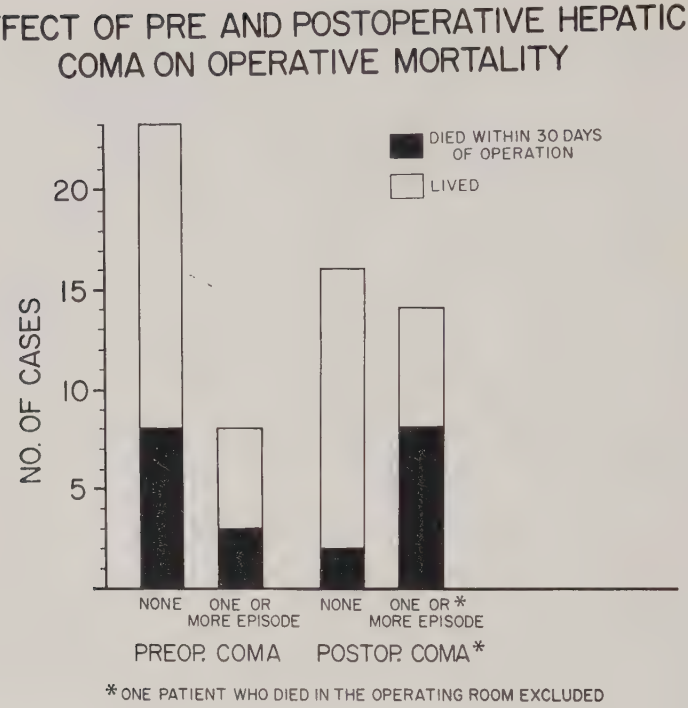

Figure 10

1.8 cm. in greatest diameter or less. In these acutely bleeding patients a large shunt seems to be the best assurance against continued or recurrent hemorrhage, and in most of our patients we have been able to construct a shunt measuring 2 cm. or more.

Preoperative and Postoperative Hepatic Coma

Hepatic coma occurred on one or more occasions preoperatively in eight of 31 patients in whom emergency portasystemic shunt was done, and it occurred in 14 patients during the postoperative period. It is an indication of poor hepatic reserve and has a poor prognosis. In spite of the prognostic implications of this event, there were six patients who experienced coma during their postoperative course, who recovered and survived the 30-day postoperative period (Fig. 10).

SUMMARY

A technique of portacaval shunt recommended for emergency control of hemorrhage from esophagogastric varices has been described. Its

principal features in the order of their accomplishment are: (1) lateral positioning of the patient on the operating table, (2) subcostal incision, (3) exposure of the portal vein through an incision on the superior margin of the foramen of Winslow, (4) avoidance of the lymphoid tissue adjacent to the portal vein, (5) exposure of the vena cava, (6) retraction anteriorly rather than resection or wedging of the caudate lobe if the lobe is in the way and (7) construction of an end-to-side portacaval shunt 2 cm. or more in greatest diameter.

Best results were obtained in patients with good hepatic reserve in whom a large shunt was constructed.

REFERENCES

1. Blakemore, A. H. and Lord, J. W., Jr.: Technic of using vitallium tubes in establishing portacaval shunts for portal hypertension. Ann. Surg. *122:* 476, 1945.
2. Child, C. G., III: The Liver and Portal Hypertension. Philadelphia, W. B. Saunders Co., 1964.
3. Eck, N. V.: On the question of ligature of the portal vein. Voyenno-Med. *130:* 1, 1877. (Translation by Child, C. G., III.): Surg. Gynec. & Obst. *96:* 375, 1953.
4. Whipple, A. O.: The problem of portal hypertension in relation to the hepatosplenopathies. Ann. Surg. *122:* 149, 1945.

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Internal Instrumentation in the Treatment of Scoliosis

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The purpose of this communication is to review methods of internal correction and fixation for scoliosis and to relate our experience with the Harrington method, to outline some of the complications we have encountered, and to define our current concepts concerning the indications and contraindications for the method.

REVIEW OF METHODS

In 1955, Allen[1, 2, 3] reported a technique for correcting scoliosis involving a distracting device applied directly to the transverse processes of the vertebrae. The instrument consisted of a turnbuckle with two Y-shaped pieces angled 30 degrees at either end. The notches of the Y's were placed against the bases of the transverse processes at either end of the concave side of the curve. The curve was then corrected by opening the turnbuckle. An arthrodesis of the spine was performed either simultaneously or one to two weeks later. No external fixation or support

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was used. The patients were allowed to sit up after two or three days and to ambulate soon afterwards. In the original series of 11 cases, four paralytic, four idiopathic, and three congenital scolioses, the initial curve averaged 94.6 degrees. The mean correction obtained was 26 degrees. The only complication reported was one instance of pneumothorax. No mention was made of infection, pseudarthrosis, or follow-up time. This was presented as a preliminary report.

In 1958, Gruca[4] reported another method of treatment based on the theory that idiopathic scoliosis is the result of muscle imbalance at the level of the primary curve. He attempted to correct this by transplanting the muscles of the scapula or arm and also by use of springs attached to the transverse processes of the vertebrae at either end of the convex side of the primary curve. The tension and the reserve left in the springs were still being studied at the time of the report, but the aim was to have the tension in the spring equal to the weight of that portion of the body above the apex of the curve. In a series of 18 patients with fixed curves of less than 30 degrees, Gruca was able to obtain correction ranging from 9.5 to 100 per cent (a mean of 50 per cent). In curves greater than 30 degrees, his results were poor and the technique was subsequently modified: first the curve was corrected to 30 degrees by a turnbuckle cast or by a device similar to Allen's and then the springs were used. Gruca's distracting device had a Y-shaped piece on either end that hooked into the transverse processes on the concave side of the curve, but distraction was achieved by means of a spring rather than a turnbuckle. Using this method in 16 patients with curves greater than 30 degrees, he was able to obtain an average correction of 36 per cent. Twelve of the patients had a second operation for insertion of springs on the convex side together with wedge resection of the vertebral bodies and intervertebral disks. Most of the patients were still under treatment at the time of the report, and could not be evaluated properly. However, in five cases with curves greater than 60 degrees, the author obtained corrections from 22.5 to 45 per cent.

In 1961, Wenger[7] reported still another method for correcting scoliosis by instrumentation using a turnbuckle similar to Allen's, but a screw was inserted into the vertebral body at each end of the curve. The surgical approach involved a paravertebral incision along the entire length of the primary curve. The medial 2 inches of each rib and the adjacent transverse processes were resected. The pleura was pushed to one side and the aorta and vena cava retracted. Screws were placed into each of the end vertebrae, the turnbuckle attached to the ends of the screws and the spine wedged open. Fusion was not done, and no external support was used postoperatively. Between 1953 and 1958, Wenger operated on 36 patients, ranging in age from 10 to 39 years. There were nine cases

with residual poliomyelitis, 25 idiopathic cases and two patients with hemivertebrae. The curves ranged from 33 to 100 degrees and the correction varied from 0 to 55 degrees. (The average correction obtained was 34 per cent.) Correction was measured early following surgery and no statement was made regarding loss of correction or follow-up time. The complications included one death due to sudden massive hemorrhage on the sixteenth postoperative day, and one case of flaccid paralysis of both lower extremities.

HARRINGTON'S METHOD

In 1962, Paul R. Harrington[5] presented a preliminary report on the correction and internal fixation of scoliosis with a new set of instruments. Since this first report, there have been many changes in the technique of application and in the design of the instruments. At present the device consists of a distraction rod for the concave side of the curve, a compression rod for the convex side, and a sacral bar that can be used for supplementary stability when necessary.

Since Harrington began using the rods in 1949, the number of complications have steadily decreased and the percentage of good results has increased. Between 1949 and 1954, he operated on 19 patients, 14 of them with residual poliomyelitis and five with idiopathic scoliosis. None was placed in a cast postoperatively. In 12, an attempt was made at operation to fuse the spine and of these, four developed pseudarthroses and in seven the rods broke.

The second group reported on by Harrington consisted of 42 patients treated between 1955 and 1960; five of these had idiopathic scoliosis and 41 postpoliomyelitic curves. Casts were not used postoperatively. In 30 of the patients, spinal fusion was carried out and only three developed pseudarthroses. Other reported complications were bone erosion in 22 or 53 per cent and instrument failure in 14 or 33 per cent.

The third group of 68 patients were all treated in 1960; 34 postpoliomyelitic scolioses, 17 idiopathic, and 17 others. In this series, spinal fusion was carried out in a larger number of patients at the initial operation –59 of 68 patients. At the same time, Harrington began to use casts postoperatively. There were only three pseudarthroses in the group, all in patients who did not have casts applied postoperatively. Of 24 patients who underwent fusion and had casts applied, there were no pseudarthroses. Other complications were instrument failure in six patients (two of these were in patients in casts), bone erosion in four patients, instrument dislocation in six patients, and infection in two patients.

Harrington does not mention the amount of correction obtained

Table 1. *Summary of Harrington's Published Results*

GROUP	FUSION (cases)	NO FUSION (cases)	INSTRUMENT FAILURE (per cent)	EROSION (per cent)	BETTER (per cent)	SAME (per cent)	WORSE (per cent)
I 1949–1954	12	5	58	52	58	26	16
II 1955–1960	30	12	33	52	59	21	20
III 1960	58	10	3	6	84	13	3

but records his results as better, the same, or worse. Patients were considered better if the curve was decreased or progression of the curve arrested and if fatigue, pain and/or respiratory distress was improved. The patient was considered the same if the curve was decreased or arrested, but there was no improvement in function. The patient was rated as worse if secondary curves developed requiring more surgery, if there was failure of the apparatus, bone erosion or progression of the secondary curves. His results are summarized in Table 1.

The only significant difference between the first two groups lies in the incidence of instrument failure. In the third group, there was also a continued decrease in instrument failure and bone erosion and improvement in end results.

At present, Harrington applies a body cast postoperatively in all cases and keeps all patients recumbent for six to eight weeks. The patients are then allowed to stand and walk for short periods, gradually increasing their activity over the next one or two months. If x-rays at that time show a loss of more than 10 degrees correction, the cast is left on for an additional one to three months depending on the age of the patient.

In 1964, Tambornino, Amburst and Moe[6] compared the Harrington instrumentation with cast correction for the treatment of scoliosis and concluded that the average correction and average loss of correction were the same for both methods. In all items, Harrington's instrumentation and cast correction were similar except for the number of infections and the number of reoperations: there was a higher incidence of infection with instrumentation but a lower incidence of reoperations.

Review of Cases

Since October 1960, 24 cases of scoliosis have been treated with the Harrington apparatus at the University of Illinois Research and Educational Hospitals. The results are similar to Harrington's first series.

There were 4 males and 17 females. The ages ranged from eight years and three months to 22 years and three months. There were 14 right dorsal, 3 left lumbar, and 4 collapsing curves in this study. The

etiologic factors were as follows: 11 postpoliomyelitic, 7 idiopathic, 1 syringomyelia, 1 neurofibromatosis and 1 muscle disease of unknown origin, presumably Werdnig-Hoffmann disease. Follow-up ranged from nine months to four and a half years. Two patients were lost to follow-up. Degrees of improvement were determined by comparing the preoperative roentgenogram with the immediate postoperative roentgenogram. Correction of the curves ranged from 60 degrees to 7 degrees. Loss of correction was determined by comparing the last follow-up roentgenogram with the immediate postoperative roentgenogram and ranged from a high of 55 degrees to a low of 0 degrees. However, in patients in whom the postoperative course was not complicated by infection or breaking of the distraction rod, loss of correction ranged from 0 degrees to 18 degrees. All patients were kept recumbent in a cast for three months following surgery except one who was kept recumbent in a Milwaukee brace. In all patients except one, arthrodesis of the spine was carried out at the time of surgery. The one exception was a "spine-jacking" procedure employed in a nine year old postpoliomyelitic boy with a collapsing scoliosis. At the time of original surgery, a long distraction rod was used to allow subsequent distraction or "jacking" of the spine as the child grew. This patient had two "jackings" before spinal arthrodesis was carried out. Of interest was the fact that, at the time of spinal arthrodesis, there was a substantial fusion mass.

In carrying out fusion of the spine, local bone was used except in one case in which heterogenous bone was employed. In the series of 24 cases there were 16 complications, summarized in Table 2.

All of the instrument failures and all cases of bone erosion occurred in postpoliomyelitic patients.

Three examples will be cited to illustrate some of the difficulties encountered, followed by two case reports to illustrate results obtained by use of the Harrington apparatus which would otherwise have been difficult to achieve.

Case I. R.K. (Fig. 1). This 12 2/12 year old girl had poliomyelitis at age 8, with subsequent development of a progressive right dorsal scoliosis. This measured preoperatively 90 degrees from D6 to L2 (Fig 1, *1*, *2*), and immediately postoperatively the roentgenogram revealed a curve of 75 degrees. Note correction of

Table 2. *Complications of Spinal Instrumentation*

A. Three infections with loss of correction after instrument removal
B. Six instrument failures (2 in 1 patient)
C. Five cases of bone erosion with loss of correction of 15 degrees or more
D. One progression of the curve below the level of the instrument
E. One case in which only 7 degrees of correction were obtained with subsequent loss of 5 degrees

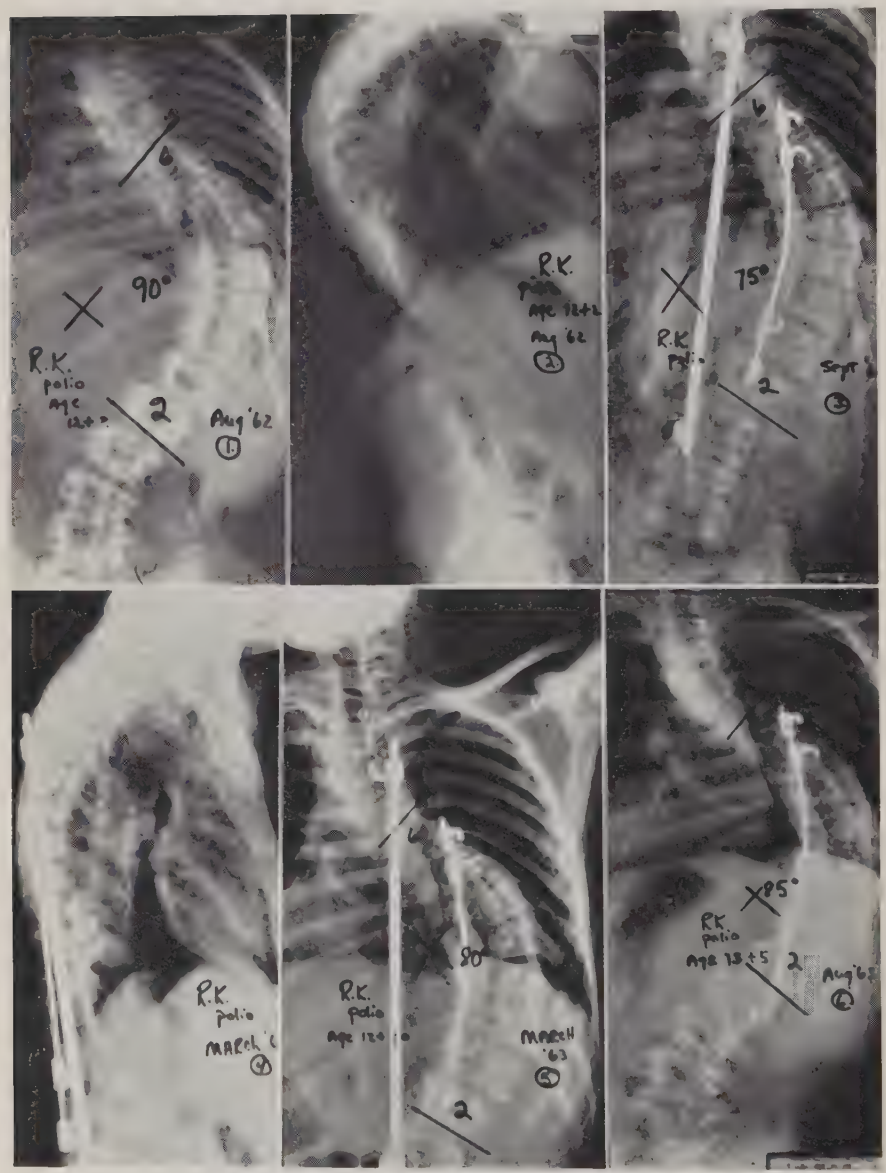

Figure 1 (Case I). For details, see text.

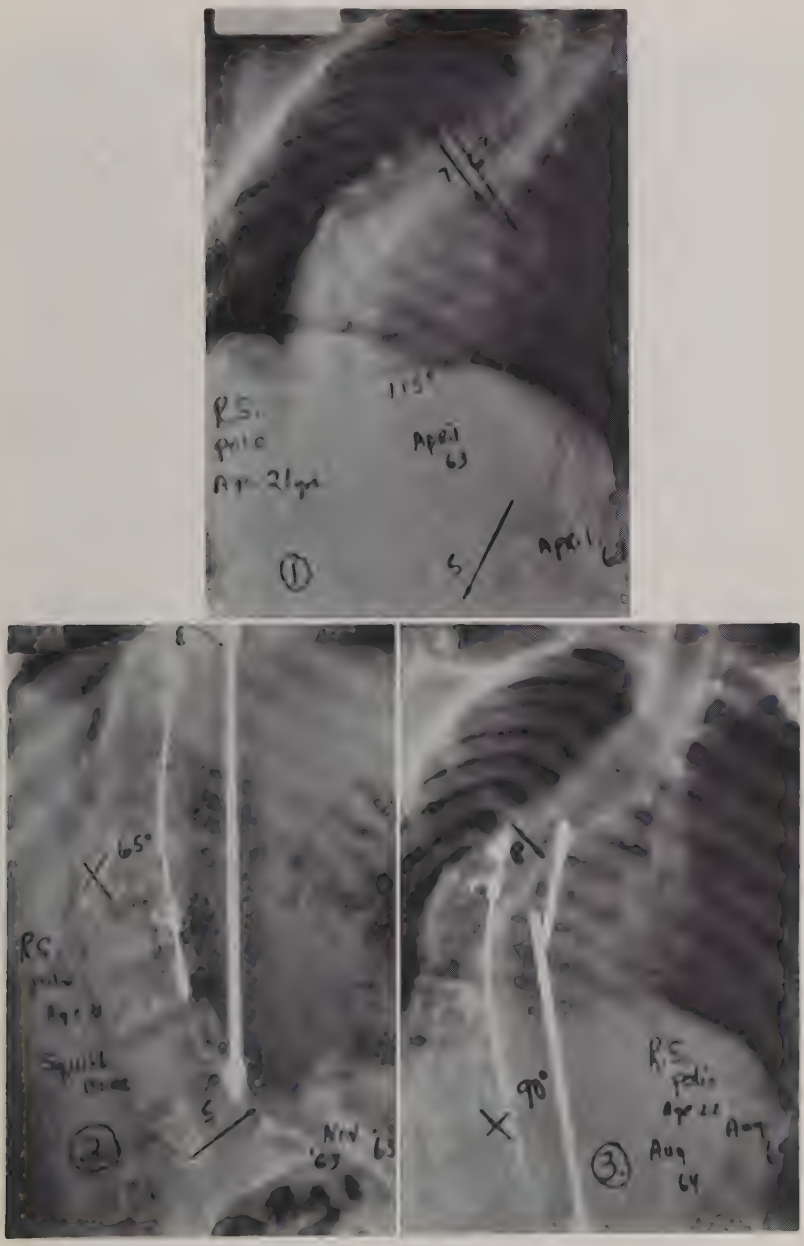

Figure 2 (Case II). For details, see text.

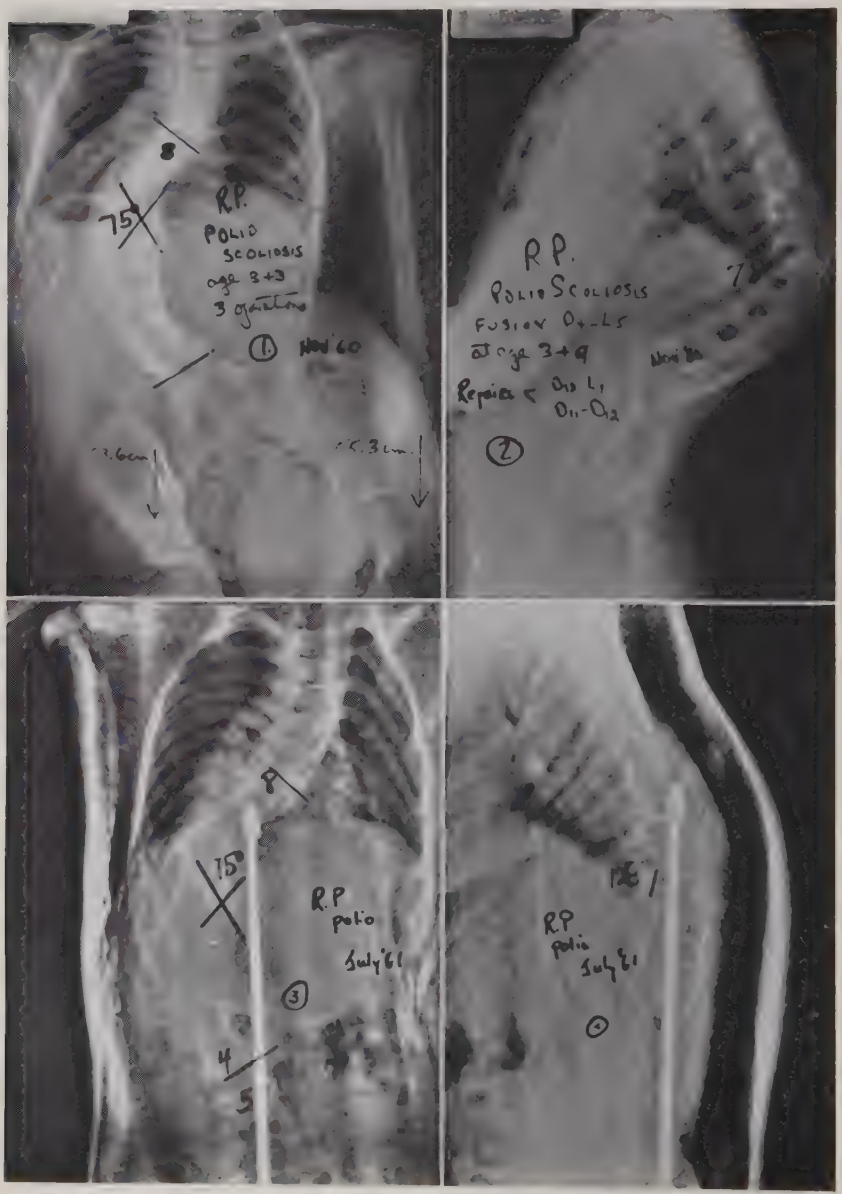

Figure 3 (Case III). See continuation on facing page and text for details.

Figure 3 (Case III). (*Continued.*)

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the kyphosis (Fig. 1, *3*, *4*). Six months later, the curve measured 80 degrees (Fig. 1, *5*), and eleven months postoperatively 85 degrees. The area of fusion was explored, no pseudarthrosis was found, but the distraction hooks were found to have torn through the soft bone. The rod was removed and the curve remained at 85 degrees (Fig. 1, *6*).

This case illustrates migration of the hooks through the soft bone, a condition termed by Harrington "bone erosion."

CASE II. R.S. (Fig. 2). This 21 year old patient had poliomyelitis at an early age and presented with a collapsing scoliosis. Preoperatively, his curve measured 115 degrees (Fig. 2, *1*). The curve was corrected at operation to 65 degrees (Fig. 2, *2*). Postoperatively, the patient was maintained in a body cast for 3 months. Fifteen months from the time of surgery, the distraction rod failed and most of the correction was lost (Fig. 2, *3*).

Instruments alone will not support a collapsing spine. The external support should have been used for a longer period of time.

CASE III. R.P. (Fig. 3). This patient with residual paralysis from poliomyelitis had had several procedures carried out on her spine. At 8 years and 3 months of age, she presented with a curve of 75 degrees. Arthrodesis of the spine had been attempted previously (Fig. 3, *1*). There was a marked kyphotic deformity (Fig. 3, *2*). Following osteotomy of the spine and attempted correction of the deformity with the internal instruments, the curve on the anteroposterior roentgenogram was not appreciably altered (Fig. 3, *3*). However, a marked change was seen on the lateral film (Fig. 3, *4*). Postoperatively, a cast was applied for 3 months. Six months after removal of the cast, the instruments failed (Fig. 3, *5*, *6*). The patient was operated on again and this time a longer period of cast immobilization was used with a satisfactory end result (Fig. 3, *7*, *8*).

Adequate immobilization should have been maintained in this case until the fusion was mature and solid. The Harrington apparatus was useful in this patient in achieving correction of a kyphoscoliosis, just as in Case I.

CASE IV. M.W. (Fig. 4). This patient had poliomyelitis (Fig. 4, *1*, *2*), and was treated initially with the halo apparatus and bilateral femoral pins to correct the spinal curve. Following correction, surgery was performed and the alignment of the spine maintained by the internal instruments as well as by the halo-pin traction fixation for 6 months. The result was satisfactory (Fig. 4, *3*, *4*).

This case illustrates how supplementary immobilization for an adequate period of time will insure maintenance of correction. In addition, the preoperative distraction was extremely helpful in reducing pressure on the soft bone, thereby preventing possible bone erosion.

CASE V. G.B. (Fig. 5). This girl had a progressive idiopathic scoliosis first noticed at the age of 5 years (Fig. 5, *1*). Spinal fusion was carried out (Fig. 5, *2*), but 5 years later the curve progressed to 65 degrees (Fig. 5, *3*) from an initial correction of 15 degrees. Osteotomy of the area of fusion and correction with the Harrington equipment were followed by a satisfactory early result (Fig. 5, *4*). The patient

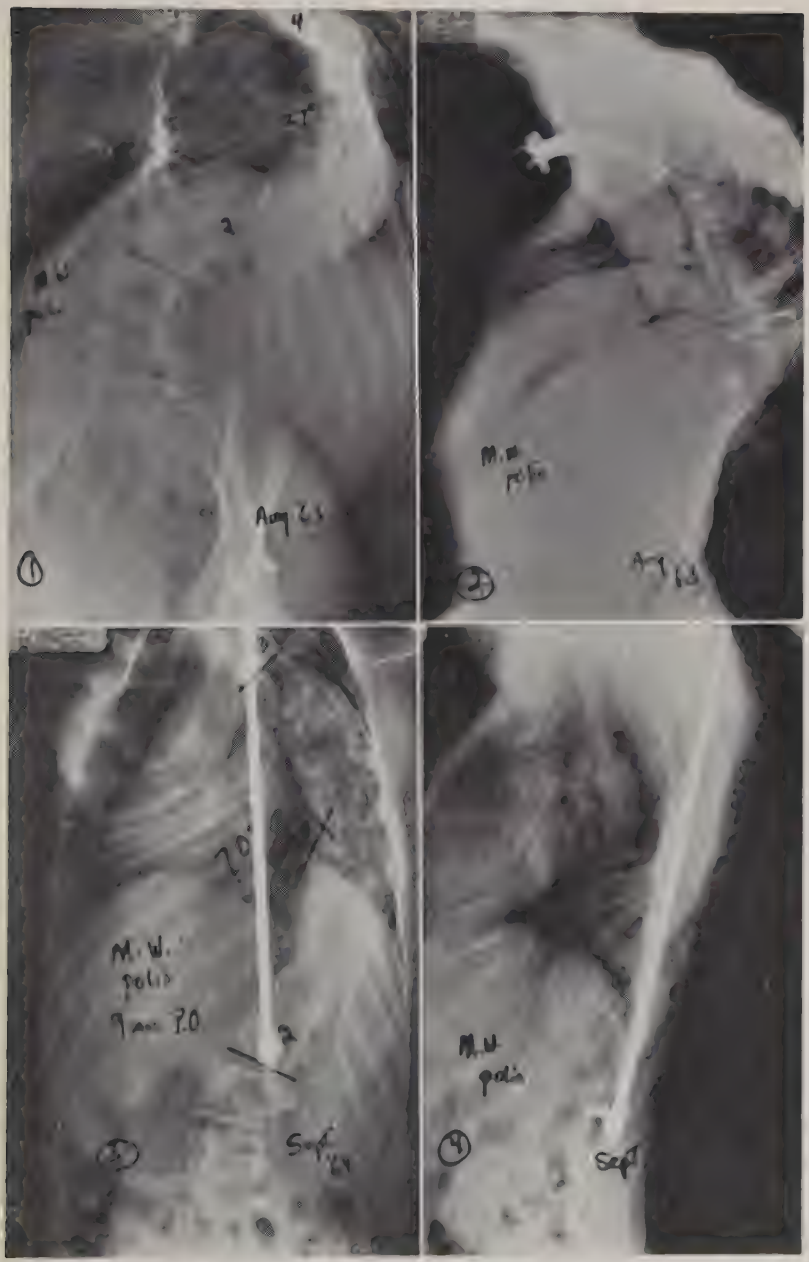

Figure 4 (Case IV). For details, see text.

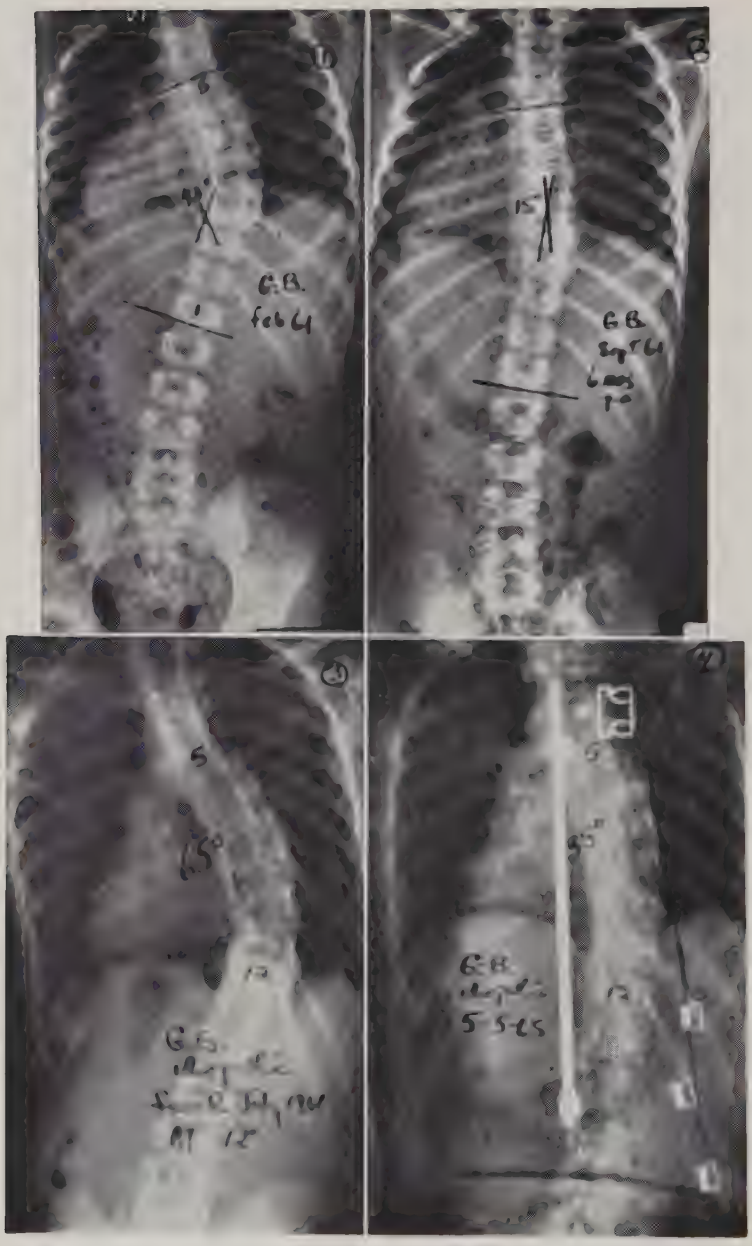

Figure 5 (Case V). For details, see text.

is now 6 months post-surgery and is being maintained in a supine position in a plaster cast extending from the chin-occiput to the knees.

SUMMARY AND CONCLUSIONS

The literature on spine instrumentation and the experience of the Orthopedic Department at the University of Illinois Research and Educational Hospitals with this equipment in the treatment of scoliosis have been reviewed.

Apparently, there are no contraindications to the use of the equipment if it is remembered that soft osteoporotic bone will not accept the distraction forces and that additional adequate external support must be used postoperatively. Preoperatively the scoliosis should be corrected and postoperatively the correction should be maintained with halo-femoral pin fixation, a localizer cast, or the Milwaukee brace until the spinal fusion is solid and mature.

Spinal instrumentation is not used routinely at present but is preferred for those cases which cannot be treated by classical methods. This is particularly true in patients with kyphoscoliosis where two plane correction can be more readily accomplished, and in patients with arthrodesis of the spine that requires osteotomy for further correction.

REFERENCES

1. Allen, F. G.: Scoliosis, operative correction of fixed curves. J. Bone & Joint Surg. *36B:* 145, 1954.
2. Allen, F. G.: Scoliosis, operative correction. J. Bone & Joint Surg. *36B:* 687, 1954.
3. Allen, F. G.: Scoliosis, operative correction of fixed curves. J. Bone & Joint Surg. *37B:* 92, 1955.
4. Gruca, A.: Pathogenesis and treatment of idiopathic scoliosis. J. Bone & Joint Surg. *40A:* 570, 1958.
5. Harrington, P. R.: Treatment of scoliosis. Correction and internal fixation by spine instrumentation. J. Bone & Joint Surg. *44A:* 591, 1962.
6. Tambornino, J. M., Amburst, E. N. and Moe, J. H.: Harrington instrumentation in correction of scoliosis: A comparison with cast correction. J. Bone & Joint Surg. *46A:* 313, 1964.
7. Wenger, H. L.: Spine jack operation in the correction of scoliotic deformity. Arch. Surg. *83:* 123, 1961.
8. Perry, J. and Nickel, V. L.: Total cervical spine fusion for neck paralysis. J. Bone & Joint Surg. *41A:* 37–59, 1959.

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Present Status of Biliary Tract Surgery

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Operations upon the biliary tract are among the procedures most frequently performed in general hospitals, and with the increase in population are being performed more often than ever before. The majority of these are performed for gallstone disease. During the past two or three decades, there has been a reduction in the mortality and morbidity associated with uncomplicated biliary tract surgery. However, diagnostic problems, complicated cases, and operations in the older age group continue to challenge the surgeon.

DIAGNOSTIC METHODS

The surgical approach to biliary tract disease has been aided greatly by the development and refinement in radiologic methods which have been used in diagnosis. These have increased the accuracy of diagnosis and have facilitated the early detection of disease. In the 40 years that have elapsed since the discovery of cholecystography by Graham and Cole, there has been a progressive improvement in the radiologic measures for the detection and estimation of the extent of biliary tract disease.

Oral Cholecystography and Intravenous Cholangiography

Oral cholecystography and intravenous cholangiography are satisfactory in the majority of patients. The intravenous method is primarily of value in patients who have had a cholecystectomy or in whom oral administration of medication is not desirable or is contraindicated. Thus intravenous cholangiography is useful in patients who continue to have symptoms after cholecystectomy, who are suspected of having common duct calculi or cystic duct remnants. Also, the procedure is helpful in the diagnosis of acute cholecystitis. The gallbladder will not visualize

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in patients with an acute inflammatory process of the gallbladder because the cystic duct is obstructed. If visualization of the gallbladder is achieved, the patient is suffering from some process other than acute cholecystitis. Except in some instances of hemolytic jaundice, neither oral nor intravenous methods are successful in patients with serum bilirubin levels greater than 3 mg. per 100 ml. Percutaneous cholangiography has attracted increasing attention more recently because of its contribution in patients with jaundice, particularly when the usual oral or intravenous methods cannot be used.

Percutaneous Transhepatic Cholangiography

Percutaneous transhepatic cholangiography had been reported a few times since the initial description of Burkhardt and Müller,[1] but its potential usefulness became apparent with the more general use of the improved radiologic techniques which employ the image amplifier and television monitor. The striking advantage of percutaneous cholangiography is the clarity with which it outlines the biliary tract and defines the nature and site of obstruction, when present.[2]

A brief description of the procedure and the principles involved is warranted. If the proper safeguards are taken, this method is safe, although reference to the earlier experience clearly indicates that potential dangers are present. The patient is prepared as though a laparotomy is to be performed. If evidence of cholangitis has been present, tetracycline is administered for 24 hours. Premedication of meperidine (Demerol) and phenobarbital is given to the patient before he is taken to the radiology unit. When the patient has been placed on the x-ray table, the skin of the abdomen is suitably prepared and drapes are applied. The skin in the midclavicular line is infiltrated with a local anesthetic agent, approximately 3 cm. below the costal margin. A needle, No. 19-21, 6 inches long, is inserted through the abdominal wall into the substance of the liver and is directed upward and medially. When bile is encountered, a sample is obtained for culture and 50 per cent sodium diatrizoate (Hypaque) is injected. From 20 to 60 ml. of the contrast agent are required, depending upon the dilatation of the biliary tree. The process of injection is facilitated by use of the television monitor and appropriate films are obtained during the procedure.

If bile is not encountered after reasonable manipulation of the needle, slow advancement of the needle within the substance of liver is accompanied by injection of small quantities of contrast agent. Occasionally, a duct can be detected by this technique and a successful study obtained. However, it has become apparent that failure to cannulate the ductal system is strong evidence that the biliary tree is not obstructed.

When a successful examination has been performed and a dilated

Table 1. *Percutaneous Transhepatic Cholangiography*

DIAGNOSIS	NUMBER
Carcinoma of pancreas	10
Choledocholithiasis	7
Carcinoma of common duct	1
Stricture of common duct	1
Hepatic metastases	1
Parenchymal hepatic disease	3
TOTAL	23

biliary tract has been visualized, the bile, containing the contrast agent, is aspirated as completely as possible, and the patient is taken to the operating room. If the ductal system is not detected, the patient is returned to the patient's unit for careful observation with frequent recording of vital signs.

Experience with 23 examinations (Table 1) has led to the conclusion that this method is valuable in the assessment of the patient with jaundice, if operation is being considered. As with needle biopsy of the liver, leakage of bile and hemorrhage are the two complications which have been reported most often. Complications have not occurred in our series. Leakage of bile has been noted at the time of operation in most patients with obstructive jaundice. Bile often may be seen to flow from the site of needle puncture and to continue to escape until the biliary tree has been decompressed. This leakage of bile from the site of insertion of the needle has been decreased by careful aspiration of bile and radiopaque dye at completion of the examination. If decompression of the biliary tract cannot be achieved, drainage of the site of liver puncture is required.

It seems likely that percutaneous cholangiography will occupy an important place in the diagnosis of patients with jaundice. In most patients with obstruction of the biliary tract, it is possible to determine accurately the location and nature of the obstructing lesion before operation is performed (Fig. 1). Failure to obtain cholangiograms correlates well with the presence of hepatitis, cirrhosis, or other parenchymal disease. The hazards should be no greater and perhaps less than the frequently performed needle biopsy of the liver.

Needle Cholangiography

Needle cholangiography during operations for gallstone diseases has continued to occupy an uncertain status in the minds of many. When cautiously utilized, it can be a valuable aid in determining whether the common duct should be explored.[5] Needle cholangiography should be considered in patients with gallstone disease who have a recent history

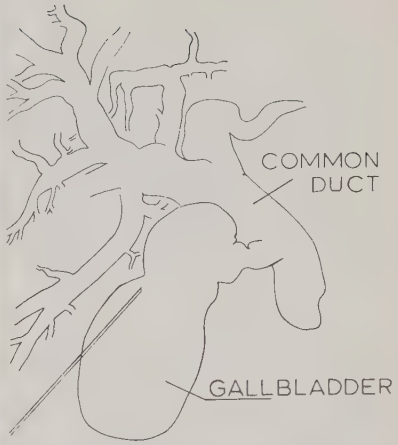

Figure 1. Percutaneous cholangiogram demonstrates marked dilatation of extrahepatic biliary tree. The rounded termination of the common duct was correctly interpreted as carcinoma of pancreas.

of jaundice but in whom stones are not felt within the common duct at the time of operation. A dilated common duct without palpable calculi is another indication for its use. The procedure may contribute to the management of the patient who has multiple small stones in the gallbladder, especially in those with large cystic ducts.

The use of this method presumes that x-ray facilities are available in the operating room and that the patient is placed on an operating table which has an x-ray cassette holder.

Puncture of the common duct is made with a fine calibre needle, No. 23 or 25, and a sample of bile is obtained for culture. The needle is attached to a segment of venatube and a small quantity of 20 per cent Hypaque, 3 to 5 ml., is injected. One exposure is made and additional contrast agent is injected, following which additional films are obtained. If a stone or other obstructing lesions are found, common duct exploration is undertaken. If there is absence of obstruction, as much bile and contrast agent is allowed to flow from the venatube as possible. Adequate drainage is required. Bile may leak from the site of puncture for two to six days. The procedure is safe and may prevent unnecessary exploration of the common duct. However, unsuspected common duct calculi may be detected, if present, and thus choledocholithotomy undertaken.

CLINICAL PROBLEMS

The complexity of certain problems which arise within the biliary tract remains challenging. Although diagnostic methods have improved, infection has been controlled better, and bleeding has become less haz-

ardous, satisfactory solutions have not been found for several clinical problems. Perhaps most serious and most difficult is the correction of common duct stricture.

Stricture of the Common Duct Due to Operative Injury

The most common cause of stricture is damage to the common bile duct during cholecystectomy. Failure to identify normal structures properly or to recognize anatomical variations seems to be responsible for most injuries to the extrahepatic biliary tree during cholecystectomy. At times, hemorrhage has occurred and the common duct has been injured during control of bleeding. Careful identification of the structures forming the triangle of Calot is the most important step in avoiding common duct injury.

Proper emphasis must be placed on prevention of injury to the common duct during cholecystectomy, because repair of the injured common duct remains a serious technical problem. Although there has been discussion over the type of repair that should be undertaken after injury of the common duct, recent reports strongly support the concept of end-to-end anastomosis of the common duct as soon as possible after the injury has occurred.[3] When the distal portion of the common duct is unsuitable for anastomosis or cannot be identified, or when end-to-end anastomosis has failed, choledochojejunostomy, Roux-en-Y, is utilized. Suture of the end of the common duct remnant to the side of jejunum has provided better approximation of the mucosa.

While the consequences of transection of the common duct are well known, the results of partial removal or damage to the wall of the common duct do not seem to be as well appreciated. In some patients, years may elapse before the results of damage to the bile ducts become apparent. The following case illustrates the long interval that may pass before the consequences of injury become manifest.

H.M., a 49 year old man, was admitted to the hospital with abdominal pain intermittently present for 3 months. The pain was located in the epigastrium and right upper quadrant, radiated to the right scapular region, and was associated with fever and dark urine. Twenty-four years before admission, he had a cholecystostomy followed a few weeks later by a cholecystectomy. Recovery was uneventful and he was well until the present illness. Examination showed mild icterus. Total serum bilirubin was 5.2 mg. per 100 ml.; alkaline phosphatase was 8.8 Bodansky units; other liver function tests were normal. When operation was performed, a common duct stricture distal to the junction of the hepatic duct was found, as well as common duct stones. The stricture was removed and choledocholithotomy and an end-to-end anastomosis performed. The T-tube was left in place for 6 months and removed. The patient was well 2 years later.

While it cannot be determined with certainty, injury to the common duct must have occurred in this patient at the time of cholecystectomy 24 years earlier. More than two decades passed before the consequences

of progressive stenosis became apparent. This contention is supported by the lack of alteration of liver function other than the elevated bilirubin and alkaline phosphatase. It was concluded that a portion of the common duct wall was removed with the cystic duct when cholecystectomy was performed.

The repair of the injured common duct requires meticulous surgical technique with careful approximation of the cut ends. The technical aspects are influenced by the condition of the duct, and in the presence of marked scarring or severe inflammatory changes a less satisfactory result may be anticipated. A better result may also be expected if there is minimal, or absence of, leakage of bile from the anastomosis. The ultimate outcome is also dependent upon the degree of liver damage that may have occurred as a result of the obstruction to the biliary system. Despite optimum conditions for repair, stricture may again occur, and therefore continued observation of the patient is indicated. Determination of serum bilirubin and alkaline phosphatase should be performed at six-month intervals for three to five years after repair of the common duct. Intravenous cholangiography is helpful in following the status of the common duct but need not be performed as often as the blood tests.

The uncertain results of common duct anastomosis and the development of stenosis with end-to-end repair or with choledochoenteric anastomoses has led to the use of various tubes and tissues to bridge gaps in the common duct.[6, 8] As yet, these have not been demonstrated to be uniformly successful, although encouraging experimental results by some investigators merit further study.

Malignant Stricture of the Common Duct

Not all strictures of the common bile duct are benign in nature. The infrequency with which carcinoma of the extrahepatic biliary tract is encountered contributes to the difficulty in diagnosis.[7] The following case illustrates the obscure nature of this disease and the unfavorable outlook with which it is associated.

S.R., a 51 year old woman, was admitted to the Chicago Wesley Memorial Hospital for the first time January 31, 1964. She complained of vomiting and epigastric pain of 2 days' duration. In 1961, she had acute cholecystitis for which a cholecystostomy was performed. Cholecystectomy was accomplished in 1962. In September, 1963, she developed evidence of obstruction jaundice and was subjected to laparotomy. Stenosis of the common duct was found, but common duct stones were not present. A T-tube was inserted. Recovery was slow and weight loss continued. She had intermittent chills and fever.

Two days before admission, she developed vomiting. Hepatomegaly was present and the abdomen was moderately distended. She was mildly anemic. A T-tube cholangiogram showed the common duct to be obstructed in the midportion, and

gastrointestinal roentgenograms disclosed marked gastric retention. A posterior gastrojejunostomy was performed February 7, 1964. Recovery was uneventful and the patient improved.

She was admitted again April 1, 1964, with nausea and jaundice. The liver edge was smooth, moderately tender and firm. Serum bilirubin was 2.6 mg. per 100 ml. The T-tube was found to be occluded and was removed. However, a progressive increase in icterus followed. Operation was performed April 22, 1964. Dense fibrosis was found in the region of the porta hepatis, but both left and right hepatic ducts were found. A choledochojejunostomy, Roux-en-Y, was accomplished. Surgical pathologic report from examination of tissue removed from the region of the common duct was "poorly differentiated adenocarcinoma in fibrous connective tissue, clinically bile duct." The patient recovered but was reported to have become icteric 4 months later.

This case illustrates many characteristics of carcinoma of the bile ducts. The diagnosis was not made before the last operation and apparently was not suspected at the time of T-tube insertion six months earlier. Biopsy of the common duct should be performed when a stricture is repaired. The progression of jaundice, weight loss and pain are symptoms that suggest bile duct carcinoma. The percutaneous cholangiogram was not diagnostic in this patient (Fig. 2), although the configuration of the occluded duct was compatible with the presence of neoplasm. Despite occasional encouraging results when resection can be performed, in general the prognosis is poor and death from bile duct obstruction occurs. Removal of the lesion or relief of the jaundice by decompression of the

Figure 2. The percutaneous transhepatic cholangiogram in this patient with carcinoma of the bile duct demonstrates only the right hepatic duct.

biliary tract by drainage into the gastrointestinal tract should be attempted if at all feasible.

DISCUSSION

This paper has considered the role of percutaneous cholangiography in the diagnosis of biliary tract disease and the procedure has been recommended as useful in the patient with jaundice of uncertain origin. Stricture of the common duct was selected as a serious problem, representing the complexity of certain biliary tract lesions.

Numerous other problems might be considered worthy of discussion as well. Acute cholecystitis, the most common complication of chronic gallstone disease, has been the subject of controversy in past years. More recently, the surgical approach to acute gallbladder disease seems to arouse less disagreement. While there is variation in interpretation of early operation, prompt surgical intervention is more generally recommended.

Basic to the entire topic is the cause of the development of gallstones. Although diet must be a factor, the failure to correlate gallstone formation with serum cholesterol levels or with dietary fat has been discouraging. It is apparent that more sophisticated methods will be required if the cause of cholelithiasis is to be found. Physicochemical methods have been introduced in an effort to understand the significance of colloidal aggregation or micelle formation in bile.[4] There is need for more research in this important field. Investigative activity has failed to keep pace with the clinical status of biliary disease.

REFERENCES

1. Burkhardt, H. and Müller, W.: Versuche über die Punktion der Gallenblase und ihre Roentgendarstellung. Deutsche Ztschr. f. Chir. *161:* 168, 1921.
2. Flemma, R. J., Capp, P. M. and Shingleton, W. W.: Percutaneous transhepatic cholangiography. A.M.A. Arch. Surg. *90:* 5, 1965.
3. Glenn, F.: Postoperative strictures of the extrahepatic bile ducts. Surg. Gynec. & Obst. *120:* 560, 1965.
4. Rains, A. J. H.: Researches concerning the formation of gallstones. Brit. M.J. *5306:* 685, 1962.
5. Seiro, V. and Kettunen, K.: Operative needle cholangiography in gall-stone surgery: Indications and results. Acta chir. scandinav. *129:* 96, 1965.
6. Sherman, R. T., Jackson, T. M., Nielson, C. A. and Wilson, H.: Reconstruction of the common bile duct with an acrylate-amide prosthesis. Ann. Surg. *158:* 420, 1963.
7. Strohl, E. L., Reed, W. H., Diffenbaugh, W. G. and Anderson, R. E.: Carcinoma of the bile ducts. A.M.A. Arch. Surg. *87:* 567, 1963.
8. Thomas, J. P., Metropol, H. J. and Myers, R. T.: Teflon patch graft for reconstruction of the extrahepatic bile ducts. Ann. Surg. *160:* 967, 1964.

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A Technique for Dissection of the Axilla

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Axillary dissection, as an isolated procedure, is performed for malignancies of the upper extremity and trunk. Berg and Robbins[1] advocate the dissection concurrently with simple mastectomy for poor-risk patients suffering from carcinoma of the breast. Crile[3] suggests a simple mastectomy for clinical Stage I carcinoma of the breast to be followed by a subsequent axillary dissection if evidence of axillary metastases presents. Malignant melanoma arising on the trunk or upper extremity is often treated by either elective ("prophylactic") or "therapeutic" axillary dissection combined with a wide variety of excisions of the primary tumor.

From personal experience with and evaluation of the published techniques for isolated axillary dissections,[2, 4, 5, 7, 8, 10–13] we have found certain objections to them: (1) Skin incisions are often made across the axilla. This is not cosmetically attractive as it does not lie in a natural skin fold or line and may lead to limitation of shoulder motion with healing and subsequent contracture. (2) The pectoral muscles are divided or removed. If the divided muscles are resutured, there is usually a significant loss of function due to interference with nerve and blood supply to the muscles. There is associated muscle fibrosis, and periarthritis of the shoulder often develops. (3) Except when dealing with mammary carcinoma, removal of the pectoral muscles is not usually necessary as lymphatic channels do not course through these muscles from the majority of areas of the trunk or upper extremity.

ANATOMICAL CONSIDERATIONS

Because the rationale for dissection of the axilla is basically a re-

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moval of a regional nodal basin draining the upper one-half of the body and the dissection itself is a pure anatomic exercise, it is pertinent to present some of the basic anatomic considerations pertaining to the lymph nodes of the axilla and the anatomic boundaries of the space.

Axillary Lymph Nodes

The axilla contains five principal groups of lymph nodes:

1. *Axillary Vein Group.* These lie along the lateral portion of the axillary veins usually on its caudal aspect.

2. *Scapular Group.* These lie along the subscapular blood vessels and extend from the lateral thoracic wall to the axillary vein.

3. *External Mammary Group.* These lie along the medial wall of the axilla, outside of the fascia covering the digitations of the serratus anterior muscles, extending from the sixth rib to the axillary vein and following the course of the lateral thoracic artery.

4. *Central Group.* These lie embedded in fat in the center of the axilla and constitute the largest and most readily palpable group of axillary nodes.

5. *Subclavicular Group.* These lie at the apex of the axilla and extend to the point where the axillary vein disappears beneath the subclavius muscles. Lymphatic channels from all the other groups of axillary nodes empty into the subclavicular nodes.

Lymphatic Drainage of the Trunk and Upper Extremity

Lymphatic channels from different areas of the body are compartmentalized so that the majority drain into specialized nodal regions. Operative attempts at removal of regional nodal basins then must be predicated on a knowledge of the common lymphatic pathways.

Breast. The majority of the efferent lymphatic channels of the mammary gland drain to the external mammary nodes. A few channels pass directly to the central nodes and then to the subclavicular group of nodes. Lymphatic channels from the medial portion of the breast accompany perforating branches of the internal mammary vessels to nodes of the internal mammary chain. Lymphatics penetrate the pectoralis major along the course of the thoraco-acromial artery to enter the subclavicular group of nodes.

Upper Extremity. Lymphatic channels of the arm tend to course medially toward the epitrochlear area and ascend mainly on the medial aspect of the upper arm to the axillary group of nodes. Channels originating in the upper arm also tend to course medially, and drain to the axillary glands. Some lateral superficial trunks accompany the cephalic vein; these usually empty into high axillary nodes after piercing the costocoracoid membrane, but may empty into supraclavicular nodes in

the transverse cervical chain. Lymphatics from the radial aspect of the hand may also course to the supraclavicular nodes.

Drainage from the shoulder may reach the scapular group of nodes below, or the central and high axillary group via channels accompanying the thoraco-acromial vessels. Drainage may also pass directly to the supraclavicular and deep lateral cervical chains, and to nodes accompanying the transverse scapular and transverse clavicular vessels.

Upper Trunk. Lymphatic channels from the upper trunk drain toward the axilla, and those of the lower trunk drain toward the groin. Although Rouviere describes the dividing line as running from the umbilicus to the level of the second lumbar intervertebral disk, posteriorly,[9] Taylor and Nathanson have noted lower trunk lesions with groin metastases.[13] Anterior trunk lymphatic channels drain into the external mammary group of nodes. Medially, channels drain into the internal mammary chain, and sometimes toward the opposite or both axillae. Posterior trunk lymphatic channels drain into the scapular group of nodes. Medial channels also may drain contralaterally or bilaterally.

A TECHNIQUE FOR ISOLATED AXILLARY DISSECTION

The patient is positioned and draped as for a radical mastectomy, with the arm abducted 90 degrees. The skin incision is made along the lateral border of the pectoralis major muscle, beginning slightly medial

Figure 1. Schematic representation of skin incision for axillary dissection.

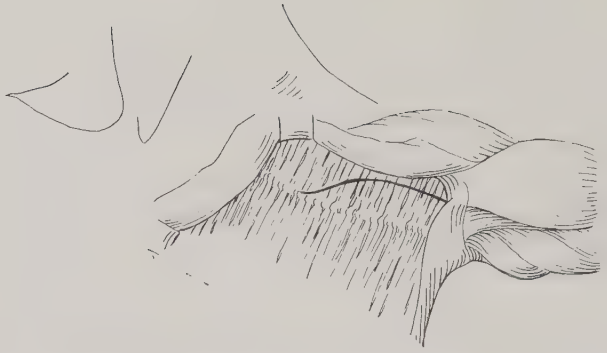

Figure 2. The skin flaps have been raised and the pectoral muscles are retracted superiorly and medially. The clavipectoral fascia is intact.

to the apex of the axilla (Fig. 1). The incision may extend to the level of the fifth through seventh rib, depending upon how thoroughly the external mammary and central nodes need to be removed. Skin flaps are raised posteriorly to the latissimus dorsi muscle, superiorly to the cephalic vein, laterally to the insertion of the pectoralis major muscle onto the bicipital groove of the humerus, and medially as dictated by the site of the lesion and whether a supraclavicular dissection is to be added to the procedure (Fig. 2). The axillary fascia is incised in the line of the skin incision at the lateral border of the pectoralis major muscle. This divides the so-called suspensory ligament of the axilla, the extension laterally of the costoclavicular fascia which invests the pectoral muscles. This maneuver frees these muscles laterally, so that they may be retracted medially and anteriorly, thus giving access to the space between the pectoral muscles and the axillary sheath. A retractor is slipped beneath the pectoralis major muscle to draw it anteriorly and medially. The clavipectoral fascia investing the pectoralis minor is incised and this muscle is also retracted upward and medially. The pectoral branch of the thoraco-acromial trunk and the medial and lateral thoracic veins are identified entering the pectoralis minor posteriorly. The vessels may be divided, but the anterior thoracic nerves should be spared, as they innervate the pectoral muscles. At the option of the surgeon, the pectoralis minor muscle may also be divided without significant disability, cosmetic deformity, or loss of function of the arm. When this operation is performed for metastatic carcinoma of the breast, dividing the pectoralis minor affords better access to the transpectoral and retropectoral lymphatic channels and to the lymph nodes of Rotter, which usually lie between the two pectoral muscles. The axilla is now open to the costocoracoid ligament medially.

Dissection of the axillary contents may be begun either medially

or laterally. We prefer to begin the dissection medially to control at the outset the efferent lymph channels. These are the subclavicular nodes; regardless of the site of the lesion they should be removed, as all other channels drain into them.

The deep pectoral fascia is incised, beginning at the costocoracoid ligament and extending laterally along the axillary vein to the tendon of the latissimus dorsi muscle. The clavipectoral fascia thus released is dissected caudad with its attached fat and areolar tissue, exposing the brachial plexus superior to the vein. The wall of the axillary vein is encountered and its adventitia incised along the anterior surface. A plane of dissection is entered which bares the vein, and enables its branches to be encountered and ligated before they are severed. The highest subclavicular nodes are dissected laterally from the recess beneath the vein where it crosses the costocoracoid ligament, and the axillary nodes are dissected out as a sheet held together by the deep pectoral fascia. In dissection for lesions of the upper extremity, the scapular nodes adjacent to the thoracodorsal nerve are usually not involved, so these nodes need not be removed and the nerve may be preserved. The long thoracic nerve is preserved if at all possible. The fascia, nodes and fibroareolar tissue are removed to about the level of the fifth rib. The resection is completed by cutting the specimen free from the edge of the latissimus dorsi muscle (Fig. 3).

"Hemovac" suction catheters are routinely used, as there is always accumulation of lymph from severed lymphatic channels. Skin flaps are closed with fine interrupted sutures. Shoulder mobilization is begun on

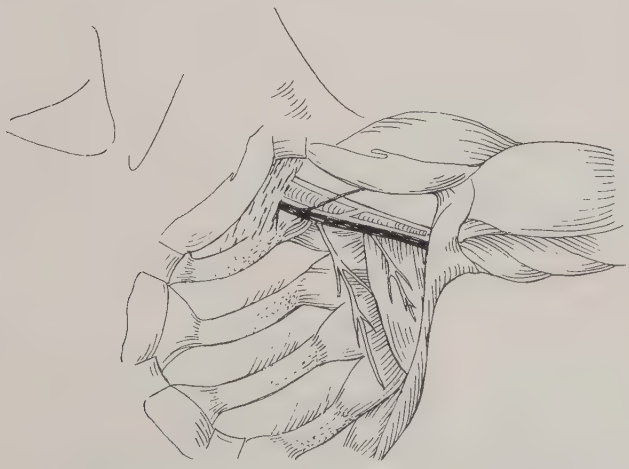

Figure 3. The dissection is completed to the level of the disappearance of the subclavian vein beneath Halsted's ligament. The thoracodorsal and the long thoracic nerves are preserved.

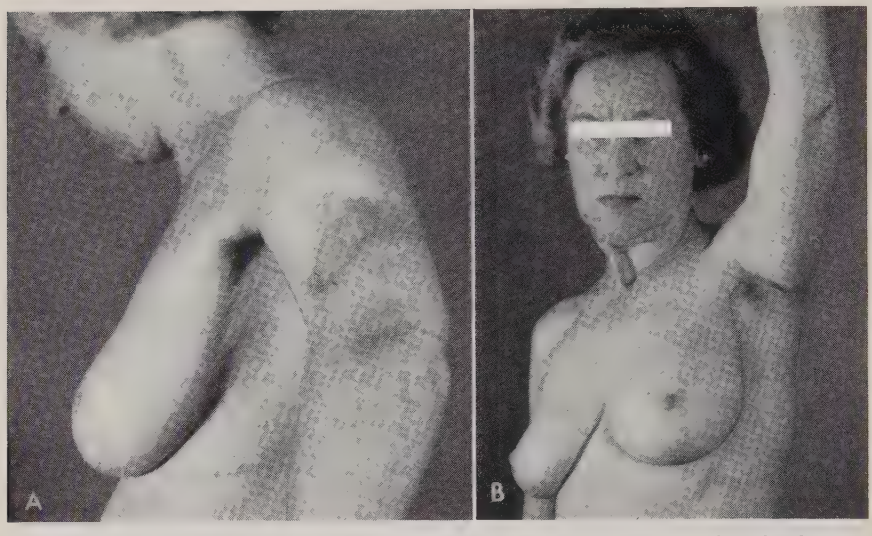

Figure 4. Mrs. A.B., a 45 year old female patient who had previous inadequate excision of malignant melanoma of the anterolateral aspect of the left arm. The previously inadequately excised tumor was widely reexcised with application of a split graft and the axilla was dissected because of palpable axillary nodes. In *A* the area of the primary excision is shown together with the depression at the axilla from the loss of axillary contents. There is minimal deformity and good range of shoulder motion as illustrated in *B*. Thirty-eight nodes were isolated from the specimen of axillary contents.

the first postoperative day, as there is the same tendency by the patient to immobilize this joint postoperatively as after radical mastectomy.

An illustrative case is presented in Figure 4.

SUMMARY

A procedure for axillary dissection is described. Location of the skin incision and preservation of the pectoralis muscles enhance the cosmetic and functional result without diminishing the thoroughness of the operation.

REFERENCES

1. Berg, J. W. and Robbins, G. F.: Modified mastectomy for older poor-risk patients Surg. Gynec. & Obst. *113:* 631–634, 1961.
2. Bowden, L.: A more thorough in-continuity neck and axillary dissection. Am. Surgeon *143:* 481–492, 1956.
3. Crile, G., Jr.: Results of simplified treatment of breast cancer. Surg. Gynec. & Obst. *118:* 517–523, 1964.

4. Frieden, J. H.: Regional lymph node dissection in cancer of the extremities. Surg. Gynec. & Obst. *89:* 591–598, 1949.
5. Gumport, S. L., Lyall, D. and Zimany, A.: A radical axillary lymph node axillary dissection for malignancy: Indications and technique. Arch. Surg. *83:* 227–230, 1961.
6. Haagensen, C. D.: A technique for radical mastectomy. Surgery *19:* 100–131, 1946.
7. Ladouz, J. E., Cernich, C. and Bracuto, D.: Axillary tumors—their approach routes. Semana méd. (Buenos Aires) *121:* 1913–1915, 1962.
8. Pack, G. T. and McGraw, T. A.: Interscapulomammothoracic amputation for malignant melanoma. Arch. Surg. *83:* 694–699, 1961.
9. Rouviere, H.: Anatomie des lymphatiques de l'homme. Paris, Masson et Cie, 1932.
10. Sedgwick, C. E.: Regional lymphatic dissection for malignancy of the extremity. S. Clin. North America *36:* 785–792, 1956.
11. Smith, J. W.: Radical axillary dissection without mastectomy. Surg. Gynec. & Obst. *100:* 245–248, 1955.
12. Spear, H. C.: Axillary gland dissection with preservation of the pectoral muscles for cancer of the upper extremity. Surgery *34:* 701–705, 1953.
13. Taylor, G. W. and Nathanson, T.: Lymph Node Metastases: Incidence and Surgical Treatment in Neoplastic Diseases. New York, Oxford University Press, 1942.

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Operation for Obstructing Duodenal Hematoma

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The relief of duodenal obstruction is among the oldest of surgical endeavors. Traumatic hematoma is one of the rarer causes of such obstructions. An apparent increase in the frequency and certain technical considerations in the management of traumatic hematomas would appear to justify their inclusion in a text on new operations.

The extravasation of blood into the layers of the duodenal wall leading to obstruction was first described by M'Lauchlan[12] in 1838, and was first considered to be a false aneurysm between the mucosa and muscularis coats. While a variety of etiologies were subsequently proposed, it was Thompson in 1855[18] who first established a traumatic basis for its occurrence. Nearly 100 years later Stirk[17] described the successful operative management of such a case. During the past ten years, a succession of case reports and clinical reviews[6, 8, 13, 14, 16, 19] support the dominant role of blunt abdominal trauma in the causation of intramural hematomas of the duodenum, and have further established their clinical and radiological features.

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PATHOGENESIS

A variety of forces, often mild in intensity, have been implicated in the development of intestinal injury. The experimental observations of Williams and Sargent[20] suggest that a shearing of the wall caught between two opposing surfaces is the primary cause of injuries due to blunt trauma. The particular susceptibility of the relatively protected duodenum almost certainly lies in its fixation and proximity to the vertebral column.[16] If at the time a blow is delivered, the liquid duodenal contents are trapped between either the pyloric sphincter, the mesenteric vessels, or the duodenal jejunal angulation, the stage is set for marked increases in intraluminal pressure, and subsequent perforation. With complete disruption of the duodenal wall, the escape of the highly irritating digestive juices proves rapidly fatal unless recognized and treated promptly. A partial disruption of the duodenal wall may occur with a far less predictable outcome.

Shearing forces which tear the rich submucosal or subserosal vascular plexuses without disrupting the mucosal and serosal coats permit the escaping blood to accumulate within the duodenal wall. Smaller collections of blood may serve only to discolor the duodenum or appear as a localized intramural tumor. While they are generally believed to subside spontaneously,[7] Altner[1] has proposed that even small hematomas may produce a cicatrizing inflammatory reaction, capable of permanently deforming the involved segment of intestine. Judd[8] and others contend that the hematoma may increase in size with the breakdown of hemoglobin within the clot. The resulting increase in osmotic pressure is believed to draw in fluid in a manner similar to that of a subdural hematoma.

Regardless of their mechanisms of origin, hematomas greater than 30 cc. in size are capable of producing obstruction of the duodenum and may impinge upon the openings of the biliary or pancreatic ducts to produce jaundice or pancreatitis. They may rupture into the free peritoneal cavity or into the retroperitoneal space, either spontaneously or as a result of operative manipulations. Since in most instances the mucosal layer of the bowel wall remains intact,[11] secondary infection or rupture into the intestinal lumen is rare.

While certain underlying disease states,[2, 10] anatomic weaknesses,[11] or disorders of the clotting mechanism[13, 15, 21] may predispose to hematoma formation elsewhere in the small intestine, they are uncommon causes of proven duodenal hematomas.[8, 19] Nevertheless, the trauma necessary to produce an intramural hematoma of the duodenum is often surprisingly slight and may go unrecognized or be easily forgotten.

Children under 12 years of age and chronic alcoholics appear particularly vulnerable to this sequel of trauma.

CLINICAL PICTURE

Two general patterns of clinical behavior are encountered. One group of patients develop signs and symptoms immediately after injury and undergo prompt surgical exploration. In this group of patients, associated injuries elsewhere in the abdomen, concomitant injury to the pancreas, or coexistent duodenal rupture may be encountered and generalizations as to treatment are difficult.

In close to 50 per cent of patients, however, a relatively asymptomatic period of one to seven days follows the initial injury. Then bile-stained vomiting and cramplike abdominal pain gradually develop and are associated with mild abdominal tenderness usually in the epigastric area. In children, in particular, an abdominal mass created by the hematoma may become palpable. Diminished intestinal peristalsis, fever and moderate abdominal distention may occur. Signs of anemia, gastrointestinal hemorrhage, obstructive jaundice, generalized peritonitis or severe pancreatitis are only occasionally encountered.

Once suspected on clinical grounds or suggested by plain films of the abdomen, the correct diagnosis can usually be made by a contrast meal examination of the upper gastrointestinal tract. The presence of partial or complete obstruction of the duodenum by an eccentric intramural mass is usually demonstrable. The involved segment usually narrows gradually and the edematous mucosa creates a "picket fence"[8] outline of contrast material. A "coiled spring" appearance arising from the crowding together of the thickened mucosal folds at the distal end of involvement is considered pathognomonic.[4]

Persistence of obstruction in spite of gastric decompression is the usual course in traumatic hematoma and laparotomy is generally required for relief.

CASE HISTORY

The following case history selected from our previously reported series of six patients[5] serves to illustrate some of the usual as well as unusual aspects of this condition.

B.J. (C.C.H. 64-45326). This 37 year old Negro male was admitted to Cook County Hospital on 11/10/64 with the chief complaint of persistent vomiting of greenish material for the past 3 days. Eight days earlier, he had undergone an

appendectomy for complaints referable to the right lower quadrant of his abdomen. A normal appendix had been removed through a small McBurney incision. Following a relatively uneventful postoperative course, he was discharged on the fourth postoperative day, afebrile, pain-free, tolerating a general diet, and having normal bowel movements. The final diagnosis on discharge was "gastroenteritis." One day after discharge, he began to vomit after meals and this persisted to the time of admission.

Past history revealed that the patient had always been a heavy drinker. He denied any recollection of recent trauma, bleeding tendencies or serious illness. He recalled that he was troubled with abdominal pain for only 24 hours prior to appendectomy and admitted to a drinking spree sometime before the pain began.

Physical examination revealed a severely dehydrated Negro male in acute distress. His blood pressure was 90/60, pulse 120, respirations 24, and rectal temperature 98.6°. Pertinent physical findings were limited to the abdomen where a slightly fluctuant but otherwise healed McBurney incision was present. The abdomen was scaphoid and nontender. No masses were palpable. Bowel sounds were hypoactive. Rectal examination revealed no abnormalities, and examination of formed stool was negative for evidences of gross or occult blood.

Pertinent laboratory findings on admission were as follows: Hematocrit, 48 per cent; hemoglobin, 14.5 gm.; white blood cell count, 15,800. Urinalysis revealed a specific gravity of 1.030, pH of 5.0, a 1+ acetone, and a trace of protein. There was no evidence of reducing substances or abnormal sediment.

Blood chemistry on admission revealed a blood urea nitrogen of 56 mg./100 ml. with a creatinine of 3.8. Blood electrolytes were: sodium, 125 mEq./L.; potassium, 4.2 mEq./L.; CO_2, 35.4 mEq./L.; and chloride, 76 mEq./L. Serum and urine amylase concentrations were within the normal rnage.

The initial impression was that of recurrent gastroenteritis and the patient was placed on nasogastric suction, with vigorous intravenous fluid and electrolyte replacement. The fluctuant area in the subcutaneous layer of the McBurney incision was aspirated and returned 10 cc. of nonclotting blood which proved sterile on culture. Complete coagulation profile revealed no evidence of abnormal bleeding tendencies.

The patient improved on management and electrolyte abnormalities returned toward normal. Efforts to reinstitute oral feedings were associated with recurrent vomiting and an upper gastrointestinal study was undertaken on the seventh hospital day (15 days postappendectomy). A near complete obstruction of the descending duodenum was noted, with retention of contrast material in the dilated stomach for over four hours. A tapering column of barium with a "coiled spring" appearance of the duodenal mucosa was noted (Fig. 1).

The following day under general anesthesia with endotracheal intubation, the abdomen was entered through a midline upper abdominal incision. Exploration revealed a massive hematoma beginning in the mid-descending portion of the duodenum and extending behind the base of the transverse mesocolon to involve the first 2 cm. of proximal jejunum (Fig. 2). The serosal surface of the duodenum was bluish-black in color, fluctuant in consistency, and had lost much of its glistening appearance.

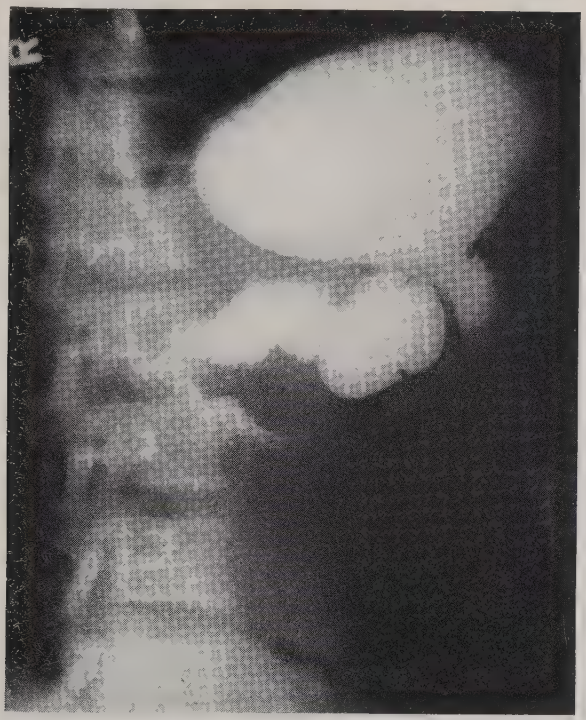

Figure 1. Upper gastrointestinal x-ray study in a patient with intramural hematoma of the duodenum demonstrating gastric dilatation, duodenal obstruction, and "coiled-spring" appearance of the duodenal mucosa.

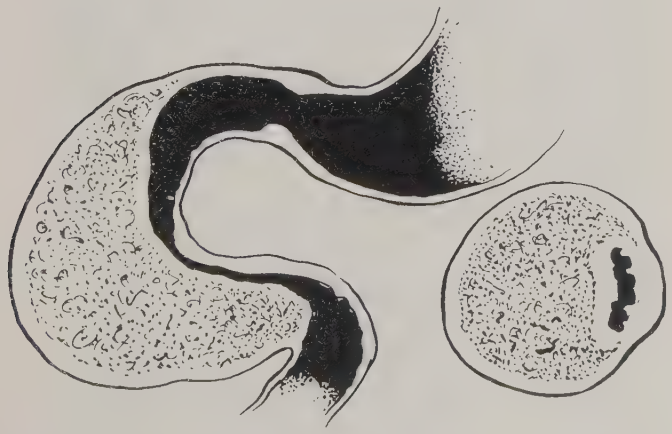

Figure 2. Artist's conception of the extensive intramural hematoma of the duodenum encountered in a 37 year old alcholic described in case report. Inset shows cross section of duodenal wall through a typical intramural hematoma.

The proximal duodenum and stomach were dilated, but the remainder of the intra-abdominal viscera, including the pancreas, biliary tract and liver, appeared normal.

During efforts to demonstrate the distal extent of the hematoma in the region of the ligament of Treitz, the serosal coat in this area ruptured with the immediate escape of approximately 200 cc. of liquid blood and partial collapse of the duodenal mass. This site of spontaneous serosal perforation was extended in a linear fashion at right angles to the long axis of the intestinal segment, and gentle compression of the hematoma was carried out. Large amounts of clotted blood (estimated 500 cc.) were evacuated by these maneuvers, which were greatly facilitated by Kocher mobilization of the duodenum. When clot evacuation appeared complete, no fresh bleeding or escaping intestinal contents were noted, and the serosal defect was closed with two layers of interrupted 4-0 black silk. Because of uncertainty as to the complete relief of duodenal obstruction and concern for the viability of the entire duodenal wall, an isoperistaltic retrocolic gastrojejunostomy was carried out and the abdomen closed.

Convalescence was complicated by evidence of efferent loop obstruction necessitating revision of the gastrojejunostomy some 20 days later. At the time of reoperation as well as during subsequent radiologic evaluations, patency of the entire duodenum was demonstrated. A functioning gastroenterostomy was also radiologically demonstrable on discharge from the hospital and the patient has returned to work free of complaints.

Comment

Although a precise history of injury was never elicited, similar experiences with other alcoholic patients have convinced us of the likelihood of a traumatic basis for this hematoma.[5] The development of a small incisional hematoma in the original appendectomy wound suggests the possibility of a clotting disorder as a contributory factor. However, on the patient's admission a coagulation profile was normal and unusual bleeding was not encountered during any of his three operative procedures.

The mistaken diagnosis of appendicitis is not uncommon in patients who do not recall an episode of trauma and serves to emphasize the frequency with which lower abdominal pain and tenderness are encountered in this condition. At the time of appendectomy, the surgeon performed a limited exploration of the abdominal cavity and observed no evidence of blood or any other abnormality in those portions of the peritoneal cavity which can be evaluated through a McBurney incision.

The relative dearth of symptoms and findings both at surgery and in the postappendectomy period serves to emphasize the subtle nature of the early state of hematoma formation. Failure to appreciate their

presence led to an early discharge from the hospital after removal of a normal appendix in spite of the magnitude of his true difficulties.

Although survey films of the abdomen may provide a valuable clue to the diagnosis, they unfortunately were not obtained prior to this patient's upper gastrointestinal examination. In several of our patients with duodenal hematomas, the gaseous distention of the stomach and proximal duodenum, obliteration of the right or both psoas shadows and a suggestion of a mass in the paraduodenal area have led to a tentative diagnosis even before contrast studies were obtained (Fig. 3).

Hematomas of the small intestine diagnosed by x-ray and treated successfully by nonoperative measures are recorded in the medical literature. It is important to point out that most such hematomas arose spontaneously in patients receiving anticoagulants, and few were located in the duodenum. In addition, most were of a small size and produced only mild symptoms of obstruction. Our own experiences leave little doubt as to the need for early surgical intervention in the vast majority of traumatic hematomas of the duodenum. Not only is prompt relief of

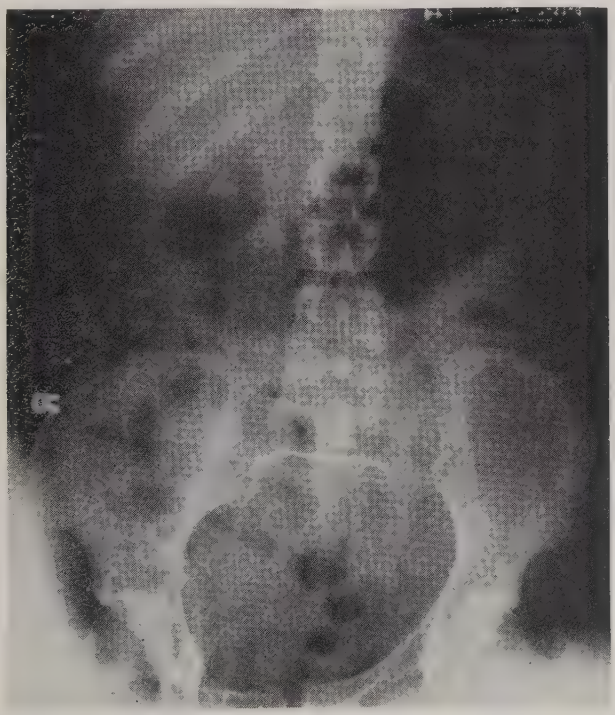

Figure 3. Photograph of a plain film of the abdomen in another patient with post-traumatic duodenal hematoma which demonstrates gastric and duodenal dilatation, obliteration of the psoas shadows, and a soft tissue "mass" in the midabdomen.

the duodenal obstruction usually necessary but some assurance of the integrity of the duodenal wall is essential. In addition, we have observed patients in whom associated damage to the pancreas and serious injuries to organs as remote as the urinary bladder have required definitive treatment along with the management of the hematoma.

THE OPERATION FOR OBSTRUCTING DUODENAL HEMATOMA

With preoperative recognition of the intramural duodenal hematoma and in the absence of other compelling indications for laparotomy, a brief period of time should be expended in evacuation and decompression of the proximal stomach and duodenum. Where barium has been used to confirm the diagnosis, this should be evacuated by the insertion of an Ewald tube and thorough lavage of the stomach. If gastric contents are largely liquid in consistency, we prefer the use of the nasogastric tube developed by Devine, which utilizes the sump principle and, in addition, provides a weighted mercury tip for intraoperative manipulations.

Simultaneous with these efforts, vigorous correction of any fluid or electrolyte deficit should be undertaken. It is seldom possible in the face of duodenal obstruction to totally correct major imbalances. Severe acid base disturbance, hypokalemia or hyponatremia, and any major reduction of the intravascular fluid compartment must be improved prior to induction of general anesthesia.

For most patients with complications of blunt abdominal trauma, we prefer a long midline incision carried well into the narrow groove between the xiphoid cartilage and costal margin superiorly and around the umbilicus inferiorly. The operation should begin with a careful and thorough exploration of the entire abdominal cavity for evidence of other sequelae of trauma or associated disease states. When attention is finally directed to the duodenum, the emphasis must be placed upon a thorough appraisal of the extent and magnitude of the injuries to the biliary, pancreatic and duodenal structures. A preliminary assessment of the extent of hematoma formation on the duodenal wall and the likelihood of complete duodenal rupture can usually be achieved without mobilization of any structures, merely by palpation and inspection of the duodenum, both above and below the root of the transverse mesocolon. It is the latter structure which holds the key to the proper evaluation of duodenal injuries. Not only does it encase essential blood vessels, such as the superior mesenteric artery and vein and its middle colic branches, but also it tends to overlie the transverse third portion of the duodenum and the entire inferior portion of the head, neck and body of the pan-

creas. Before commencing the mobilization of stomach, duodenum, pancreas or colon, it is well to elevate the transverse mesocolon and thoroughly inspect the structures about the ligament of Treitz. Not infrequently, the distal extent of hematoma formation will be found here, and this represents a frequent site of spontaneous rupture or one preferred for surgical evacuation.

In most instances, the gastrohepatic omentum is then pierced through its avascular center to enter the lesser omental bursa. The gastrocolic omentum is then incised between clamps to permit mobilization and encirclement of the distal stomach. This provides for a careful evaluation of structures which surround the lesser omental space, and the integrity and condition of the pancreas can be closely studied.

For hematomas in which the serosal layers of the duodenal wall are intact, we prefer to evacuate the hamatoma before attempting mobilization of the pancreas or duodenum. Selection of the ideal site for drainage is determined principally by its location, especially its distal extent. The general appearance and apparent viability of the tissues in the duodenal wall and the presence or absence of sites of spontaneous disruption are also considerations in the choice of the incision site.

For the usual large hematomas extending to the fourth portion of the duodenum, we prefer to incise, in a radial direction, the outer serosal layers of the wall, near the distal extent of the grossly palpable hematoma (Fig. 4). This usually results in the prompt evacuation of nonclotting blood of surprisingly large quantities, and by gentle but thorough compression of the rest of the duodenum the remaining clots can be readily extracted. Extended efforts to identify the source of hemorrhage are unnecessary as the vessels will usually have undergone thrombosis by the time surgical drainage is undertaken. Efforts to demonstrate or exclude a rupture of the duodenal mucosal coat by exploration through the site of this drainage incision is usually unwarranted.

Following as complete a removal of the hematoma contents as possible, the site of incision into the duodenal (or in some instances proximal jejunal) wall is repaired, utilizing two layers of interrupted fine silk sutures. The first of these layers attempts to reapproximate the cut edges of the outer layers of the intestinal wall and at the same time reattach the deeper submucosal structures at this site. Great care should be taken not to penetrate the mucosal layer with these sutures lest they encourage the development of a duodenal fistula. The second layer consists of reinforcing Lembert sutures of seromuscular depth, intended to approximate a broad serosal surface and encourage prompt suture line sealing. The radial direction of the incision is favored because it assures an adequate blood supply to both edges yet is unlikely to initiate further bleeding. Since the incision is at right angles to the long axis of the duo-

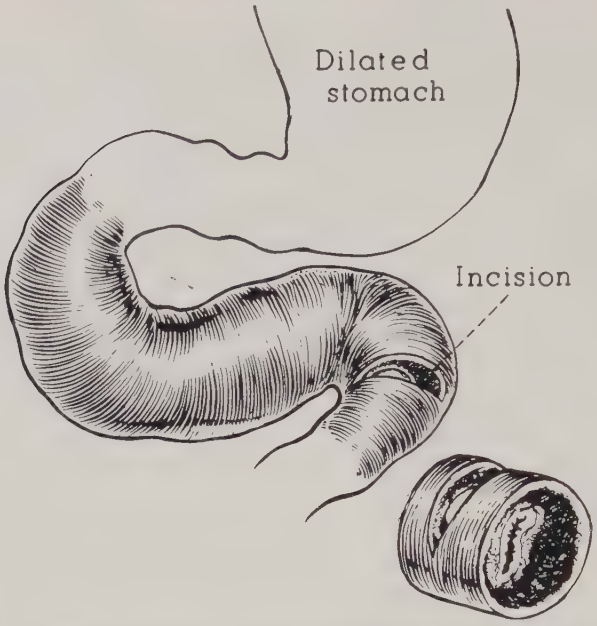

Figure 4. Artist's drawing to show preferred site of evacuation in large hematoma involving transverse duodenum and proximal jejunum.

denal lumen, even the invagination of a broad surface of distended serosa is unlikely to contribute to residual duodenal obstruction.

Following evacuation of the hematoma, any of the usual evidences of duodenal wall perforation or major pancreatic damage constitutes an indication for thorough mobilization and inspection of the entire pancreatoduodenal segment. In such cases a Kocher maneuver should be carried out and a search for evidence of escaping gas, food, blood, bile or pancreatic juice should be cautiously undertaken. The highly lethal consequences of an unrecognized duodenal leak must be weighed against the infrequency with which it is encountered in patients with the usual intramural hematoma causing duodenal obstruction. Considerable judgment and surgical skill must be exercised lest a contused duodenal wall be damaged further by these efforts. In the uncommon circumstance in which mucosal perforation accompanies duodenal hematoma, surgical identification and repair is difficult (Fig. 5). Some assurance of mucosal integrity may be achieved by injecting air or a dye, such as dilute methylene blue, down the nasogastric tube with gentle effort to demonstrate its uninterrupted passage and establish its failure to escape through the wall of the injured segment of duodenum.

In those instances in which the relief of duodenal obstruction remains uncertain or the integrity of the duodenal wall has been greatly jeopardized, we favor the creation of an antecolic isoperistaltic gastrojejunos-

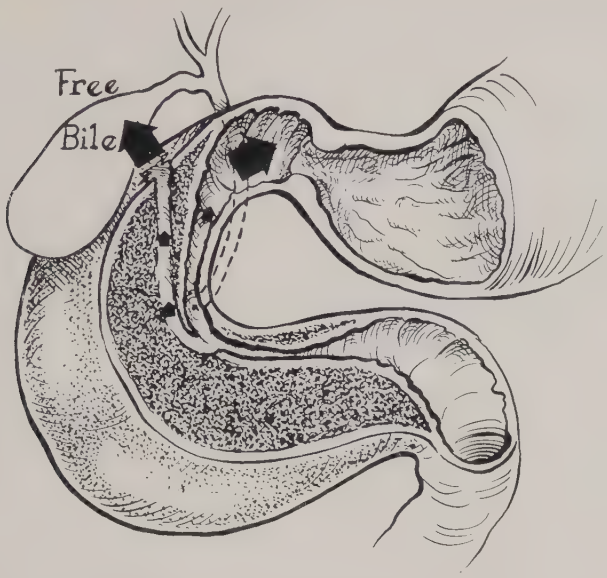

Figure 5. Artist's drawing of an unusual duodenal hematoma in which a proximal mucosal tear accompanied an extensive subserosal collection of blood and clots which drained spontaneously.

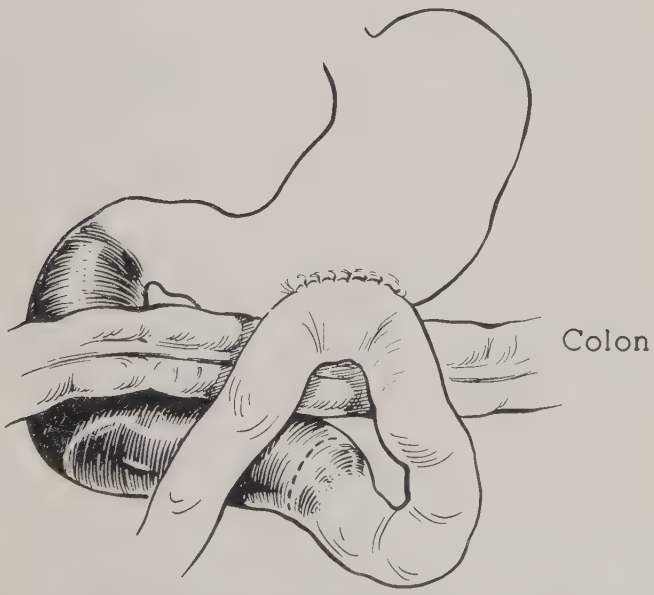

Figure 6. Artist's drawing of type of gastroenterostomy performed on patients with intramural hematomas with persistent obstruction or severe damage to duodenal wall.

tomy (Fig. 6). Besides assuring gastrointestinal continuity, additional justification for this procedure rests with its apparent effectiveness in the prevention as well as treatment of duodenal fistulas.

Our preference for an antecolic anastomosis is based upon several reports[7] and the single personal experience cited in the case report in which a posterior gastrojejunostomy functioned poorly. The difficulties which attend the creation of a window in the thickened and distorted transverse mesocolon provide a logical explanation for these difficulties. While theoretical and practical considerations of jejunal ulceration cannot be ignored, exaggerated efforts to avoid the gastrojejunostomy do not appear justifiable. The utilization of adjacent loops of jejunum for a reinforcing serosal patch is worthy of consideration when the integrity of the duodenal wall is precarious, but we have no personal experience with this in the management of duodenal hematomas. Although advocated and actually practiced by some,[13] the actual resection of segments of duodenal wall in victims of blunt trauma seems rarely if ever justified.

SUMMARY

1. A rare but challenging diagnostic and therapeutic problem occurs when blood clot fills the duodenal wall with resulting obstruction of the lumen or the ductal structures which enter it.

2. Clinical and radiologic features of the condition have been reviewed and experiences cited which suggest that traumatic duodenal hematomas with obstruction will usually require operative intervention for relief.

3. Complete evacuation of the hematoma through a carefully planned incision in the outer layers of the involved duodenal wall is the treatment of choice.

4. An anterior gastroenterostomy to bypass the area of duodenal injury should be performed whenever incomplete evacuation suggests the likelihood of continued obstruction or when the tissues which comprise the duodenal wall are of uncertain viability.

REFERENCES

1. Altner, P. C.: Constrictive lesions of the colon due to blunt trauma to the abdomen. Surg. Gynec. & Obst. *118:* 1257, 1964.
2. Bergman, B. J. and Crowson, C. N.: Intramural hematoma of the duodenum. Med. Serv. J. Canada *15:* 449, 1959.
3. Davis, D. R. and Christopher, Y. T.: Intramural hematoma of the duodenum and jejunum. Ann. Surg. *153:* 394, 1961.
4. Felson, B. and Levin, E. J.: Intramural hematoma of duodenum—Diagnostic roentgen sign. Radiology *63:* 823, 1964.

5. Freeark, R. J., Corley, R. D. and Norcross, W. J.: Intramural hematomas of the duodenum causing duodenal obstruction. (Abstr.). Proc. Inst. Med. Chicago *25:* 309, 1965.
6. Golding, M. R., DeJong, P. and Parker, J. W.: Intramural hematoma of the duodenum. Ann. Surg. *157:* 573, 1963.
7. Izant, R. J. and Drucker, W. R.: Duodenal obstruction due to intramural hematoma in children. J. Trauma, Nov., 1964.
8. Judd, D. R., Taybi, H. and King, H.: Intramural hematoma of the small bowel. Arch. Surg. *89:* 527–535, 1964.
9. Magladry, G. W., Jr. and Mathewson, C., Jr.: Duodenal obstruction due to trauma. Stanford M. Bull. *12:* 205, 1954.
10 McClelland, J. R., Thistlethwaite, J. R. and Gerwig, W. H., Jr.: Obstruction of distal duodenum due to intramural hemorrhage. Am. Surgeon *24:* 367–370, 1958.
11. Mirov, A. G.: Intramural hematoma of the duodenum. Surgery *51:* 434–436, 1962.
12. M'Lauchlan, J.: False aneurysmal tumor occupying nearly whole of duodenum. Lancet *2:* 203, 1838.
13. Moore, S. W. and Erlandson, M. E.: Intramural hematoma of the duodenum. Ann. Surg. *157:* 798, 1963.
14. Rowe, E. B., Baxter, M. R. and Rowe, C. W.: Intramural hematoma of the duodenum. Arch. Surg. *78:* 560–564, 1959.
15. Senturia, H. R., Sussman, N. and Shyken, H.: Roentgen appearance of spontaneous hematoma of the small intestine associated with anticoagulent therapy. Am. J. Roentgen. *86:* 62, 1961.
16. Spencer, R., Bateman, J. D. and Horn, P. L.: Intramural hematoma of intestine: Rare cause of intestinal obstruction. Surgery *41:* 794, 1957.
17. Stirk, D. I.: A case of subserous duodenal hematoma. Brit. M. J. *1:* 712, 1953.
18. Thompson, Alexander: Case of traumatic rupture of the duodenum. Edinburgh M. J., 1855.
19. Watne, A. L., Butz, R. and Tarnay, T. J.: Duodenal obstruction due to intramural hematoma. Arch. Surg. *89:* 441–446, 1964.
20. Williams, R. D. and Sargent, F. T.: Mechanisms of intestinal injury in trauma. J. Trauma *3:*288, 1963.
21. Wiot, J. F., Weinstein, A. S. and Felson, B.: Duodenal hematoma caused by coumarin. Am. J. Roentgen. *86:* 70, 1961.

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Recent Trends in Surgery of the Larynx

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In recent years increased interest and knowledge in the fields of laryngeal physiology and pathology have resulted in surgical developments along lines of voice preservation and vocal rehabilitation. In addition, efforts to improve the salvage rate among patients with extensive laryngeal cancers have led to the adoption of combined programs of irradiation and surgery which seem to offer great promise. The increasing incidence of laryngeal trauma in association with highway accidents has created difficult problems for the laryngologist.

GLOTTIC REHABILITATION IN VOCAL CORD PARALYSIS

In some (but not all) instances of unilateral recurrent laryngeal nerve paralysis, the involved vocal cord assumes a lateral position which interferes with closure of the glottis. The air loss which inevitably accompanies this type of paralysis is responsible for marked impairment of the voice and loss of an effective cough mechanism.

For many years various attempts have been made to increase the mass of the laterally positioned paralyzed vocal cord so that it could be met by the remaining functioning cord and glottic closure re-established. None of these efforts was consistently successful until Arnold introduced an injection technique employing a mixture of Tantalum and glycerin.[1] The method consisted of a direct laryngoscopic approach and deposition of an inert material into the paralyzed cord with a special syringe and needle. Tantalum was subsequently replaced by a Teflon-glycerin mixture. Favorable results using Teflon in a large series of patients have been reported by Lewy.[8] Untoward results have been minimal and the functional restoration of voice in individuals thus treated has been gratifying.

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Illustrative Case

CASE I. A 58 year old woman had been distressed by her weak, breathy voice since a thyroid operation 2 years previously. The inadequacy of her speaking effort had forced her to abandon her teaching position. Examination disclosed a lateral lying paralyzed left vocal cord with incompetent glottic closure (Fig. 1). Teflon injection was carried out with perceptible increase in the cord mass toward the midline and re-establishment of the glottic sphincter. Her vocal rehabilitation was complete and has remained so for 18 months (Fig. 2).

The procedure described is performed with local anesthesia and requires the use of a laryngoscope-retaining apparatus to enable the operator to use both hands for accurate placement of the injecting needle. It is necessary that the patient be awake in order to phonate during the operation, thus permitting the surgeon to judge the exact quantity needed to properly fill the cord. It is preferable to err on the side of conservatism if there is any doubt as to the adequacy of the injection. A repeat injection can easily be undertaken to correct any initial deficiency, although this is rarely required. Restoration of the voice is usually complete at the conclusion of the procedure.

CONSERVATION OF LARYNGEAL FUNCTION BY SUBTOTAL LARYNGECTOMY

The current surgical approaches to laryngeal cancer have undergone significant modifications as a result of detailed analysis of the lymphatic drainage of this structure. Whereas glottic cancer has always responded favorably to surgical measures, poorer results in the treatment of non-cordal lesions were found to be due to the presence of microscopic tumor involvement in the cervical lymph nodes.[9] Adoption of routine simultaneous neck dissection in the presence of impalpable nodal involvement has greatly improved the cure rate for lesions involving the epiglottis, ventricle or ventricular bands, arytenoids, aryepiglottic folds or pyriform sinus areas.

Consistently successful efforts to preserve laryngeal function by subtotal laryngectomy for extensive cancers previously treated by total laryngectomy were first reported from South America. The proposed two-stage procedure was modified by Ogura, who devised a unique method of extirpation and primary closure following subtotal laryngectomy and radical neck dissection.[10] This technique, which preserved both vocal and swallowing functions, is regarded as a significant milestone in the treatment of selected types of laryngeal cancer and is based upon an appreciation of the variations in lymphatic drainage of the different areas of the larynx. Contrast laryngography has been found to offer consid-

erable aid in the definition of the exact extent of these lesions since an accurate appraisal is a prerequisite for successful eradication of the neoplasm.

One of the major difficulties encountered by laryngeal surgeons in the past has been the lack of any uniformity of descriptive terminology pertaining to laryngeal cancer. In the absence of general agreement as to how laryngeal cancer should be classified, common grounds for judging the adequacy of therapy were unavailable. Fortunately many international and national groups have combined their efforts along these lines and there is now available a system of classification which will be meaningful and significant in terms of recording therapeutic results.

The American Joint Committee on Cancer Staging and End Results Reporting is sponsored by various specialty groups including surgeons, internists, pathologists, oncologists and radiologists.[2] Subcommittees composed of members and consultants with special interests and skills are active in proposing classifications for different organs and organ systems.

In the larynx, definition of anatomical regions and sites has permitted the adoption of a standard classification for the first time. Nodal and distant metastatic involvements are included in the descriptive abbreviations. When combined with clinical staging of lesions, these designations form the basis for ready appraisal in a common language. Thus, T_1 N_0 M_0 signifies a lesion confined to one anatomic site in the

Figure 1

Figure 2

Figure 1 (Case I). Mirror view of larynx showing a bowed, lateral lying left vocal cord (arrow). The right vocal cord is in the midline but cannot approximate its fellow. The widened glottis is responsible for air wastage and a hoarse, breathy voice.

Figure 2 (Case I). Mirror view of larynx of same patient after Teflon injection. The bowing has been eliminated, the cord mass has been increased and approximation of the opposite cord resulted in a good voice without air loss. The darkened color is due to hyperemia which occurs immediately after injection.

Figure 3 (Case II). Surgical specimen showing epiglottic carcinoma removed by subtotal supraglottic laryngectomy. The upper arrow denotes the superior margin of the carcinoma. The lower arrow points to the ventricular band area where the mucosa has retracted after removal of the specimen. The true vocal cords were preserved since the lesion could be excised with an adequate margin of uninvolved tissue.

larynx (T_1), without clinically palpable or suspected cervical lymph node involvement (N_0) and without distant metastasis (M_0). Varying numerical gradations are used to denote the degree of severity of the pathological processes.

Since conservation surgery is dependent upon accurate delineation of site involvement and since appraisal of any therapeutic regimen is possible only if there is uniformity of descriptive terminology, these recent developments tend to complement each other.

Illustrative Cases

CASE II. A 57 year old housewife first noted a slight sore throat 4 weeks prior to laryngeal examination. She was found to have a relatively small neoplasm (epidermoid carcinoma) involving the laryngeal surface of the epiglottis without extension to adjacent areas of the tongue or larynx and without suspected lymph node spread (Stage I, T_1 N_0M_0). The direct examination was supplemented and confirmed by cineradiography of the larynx using contrast media.

A subtotal supraglottic laryngectomy was performed with removal of epiglottis, both ventricular bands and laryngeal ventricles, preserving the true vocal cords. The specimen revealed that the primary lesion was excised with adequate margins of normal tissue (Fig. 3).

After the patient had experienced several weeks of the usual diffi-

culty in relearning how to swallow, the tracheostomy was allowed to close. Her swallowing and vocal functions were completely restored in 1 month. There has been no evidence of recurrence or nodal involvement in the 6 months following surgery.

Comment. This particular type of lesion was ideal for conservation surgery, which could not have been successfully accomplished by mere removal of the epiglottis via transhyoid pharyngotomy. Ten years ago the patient would probably have been subjected to total laryngectomy.

Case III. A 59 year old man developed hoarseness and bilateral neck swelling 2 months prior to admission. During this time another physician had obtained a biopsy specimen of the neck mass which was reported as carcinoma. Examination disclosed a huge obstructing epiglottic lesion, which was obviously the primary tumor, and bilateral fixed lymph nodes. This was classified as Stage IV, $T_2 N_2 M_0$. Only by contrast laryngography could it be determined, however, that the tumor involved the ventricular bands but not the true vocal cords.

After a short course of irradiation therapy, a supraglottic subtotal laryngectomy in continuity with a left radical neck dissection was performed. The ventricular bands and part of the ventricles were included in the resected tumor area (Fig. 4). One month later a complete right neck dissection was performed. (The neck dissections were staged because of the patient's age in an effort to avoid the complications of bilateral simultaneous removal of the jugular veins).

Figure 4 (Case III). Surgical specimen showing a large epiglottic carcinoma removed by subtotal supraglottic laryngectomy in continuity with radical neck dissection. (Neck contents not shown.) Patient has a normal voice and is well 2 years after operation.

The patient experienced some difficulty with aspiration for a short time after surgery. His swallowing function returned before the tracheostomy tube was removed. He has resumed his former job as a laborer and has remained free of any evidence of recurrence for 16 months.

Comment. Cervical adenopathy indicative of metastases may occur in over 30 per cent of patients with noncordal cancer before the primary site is recognized.[3] This is due in part to the relative lack of symptoms in the early stages of epiglottic or arytenoid area lesions and in part to their relatively rich lymphatic supply in contrast to that of the true vocal cords.

In this particular situation there was no need to sacrifice the entire larynx when surgical exploration revealed the possibility of removing the primary lesion and permitting the patient to retain his vocal cords. The validity of the concept is borne out by the fact that he has remained free of disease for 16 months following removal of the primary lesion and the bilateral lymph node chains which were the site of metastatic spread.

It cannot be emphasized too strongly, however, that the success of laryngeal surgery of any form is entirely dependent upon the proper selection of cases. One can recall that when thyrotomy and cordectomy were standard surgical procedures for vocal cord cancer, failures were usually due to attempts to accomplish more than was feasible with such a limited approach. By the same reasoning, attempts to conserve vocal function with subtotal laryngectomy should not be the primary consideration. As in all cancer surgery, cure of the basic disease is the main consideration. Any doubts as to the adequacy of the procedure should obviously be resolved in favor of a choice which will totally eradicate the neoplastic growth.

COMBINED RADIATION AND SURGERY FOR ADVANCED CANCER

Although the cure rates obtained by surgery and radiotherapy in relatively small and localized cancers of the larynx have been consistently high, extensive lesions in this area continue to pose difficult problems in management and the recurrence rate in the past has been discouraging. Frequently, a patient with a seemingly inoperable lesion was referred to the radiotherapist for palliation. Some excellent results were occasionally obtained but these were usually unpredictable and were often followed by recurrence of the neoplasm either in the original location or at a new site adjacent to it. In recent years efforts have been directed toward improving the salvage rate in extensive cancer by employing a combination of preoperative radiation and surgery.[7]

The principal aim of such preoperative radiation is to destroy the

tumor cells which are known to proliferate more rapidly at the periphery of a large neoplastic growth, to arrest growth of other remaining cells if possible and to halt tumor growth in metastatic lymph nodes.[4] Follow-up surgery is planned with full agreement between the patient, the surgeon and the radiologist that it will be undertaken despite what may appear to be an initial salutary radiation effect. Additionally, the time lapse of four to six weeks following a full course of radiation and before surgical intervention is agreed upon in advance. In an effort to determine just how much radiation is most effective in this regard, some patients have been given shorter courses of therapy and the surgery undertaken immediately.

Dr. Frank Hendrickson, Director of Radiation Therapy at Presbyterian-St. Luke's Hospital, has been following a series of patients with extensive laryngeal cancer in a program of combined radiation and surgery. Although the study is only of two years' span, the results seem to indicate that the control of extensive disease is better than by previous forms of therapy.[5] Of the 38 patients with advanced lesions followed for more than nine months post-treatment, only seven are either dead from cancer or have had recurrence of cancer. It is obvious that the interval is too short for any final conclusions but past experience has shown that most recurrences following either irradiation or surgery alone could be expected well within one year.

It has not been definitely determined whether a short or long course of radiation therapy is preferable in the preoperative period. Although a higher complication rate could be anticipated as a result of irradiation, this has not been a serious consideration in the current study and there have been no fatalities attributable to such complications.

Illustrative Case

Case IV. A 62 year old man had experienced hoarseness for 5 months, swallowing difficulty for 3 months and a painless enlargement in his right neck for 1 month. Mirror examination disclosed a fungating, fixed, obstructing neoplasm of the epiglottis. There was a firm immobile mass measuring 8 cm. in diameter beneath the angle of the mandible on the right side. Direct laryngoscopy suggested that the epiglottic lesion had invaded the ventricular bands and true vocal cords. Biopsy revealed epidermoid carcinoma. The lesion was considered to represent Stage IV, T_3 N_2 M_0 in that it involved epiglottis, ventricular bands and extended onto the true vocal cords (T_3) with clinically palpable fixed cervical lymph nodes (N_2) and no evidence of distant metastases (M_0).

A tracheostomy was performed. The patient received cobalt-60 irradiation in the dosage of 5000 r to the primary tumor area and 5000 r to each cervical lymph node chain over a period of 4 weeks. Six weeks following completion of radiation he underwent laryngectomy and right radical neck dissection (Fig. 5). Primary healing ensued and the patient was discharged 15 days after operation.

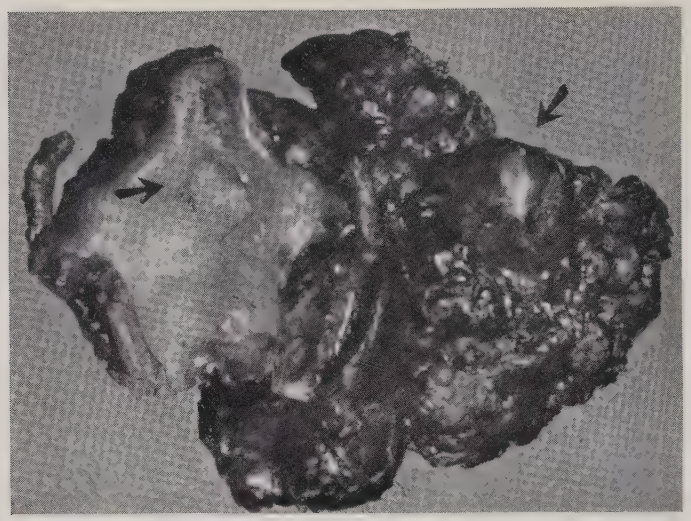

Figure 5 (Case IV). Surgical specimen showing larynx and neck contents excised in continuity following a full course of preoperative radiation. The arrow on the left designates the site of the original tumor. The arrow on the right points to a necrotic metastatic lymph node mass which had been fixed beneath the mandible prior to radiation. Patient is alive and well 2 years after operation.

The surgical specimen revealed necrotic tumor cells in the epiglottis and throughout the stroma of the larynx, including the vocal cords. The cervical lymph node mass was of creamy consistency and contained necrotic tumor cells in the depths. There were areas of recognizable tumor in the epiglottis.

The patient has remained healthy and free from recurrence for 24 months.

Comment. The result of preoperative radiation and surgery in this and similar cases is most encouraging. There seems to be no doubt that the original fixed lymph node mass was rendered resectable—and probably curable—by the preoperative radiation. Past experiences with extensive cancers of this type were uniformly discouraging and the prognosis grave. Although long-range results remain to be determined, it would seem that the maximum benefits from tumor surgery in this area have been made possible by the combined type therapy currently being employed.

LARYNGEAL TRAUMA

One of the major problems associated with automobile or industrial accidents has been the failure to recognize injuries to the larynx when concern for head, face, chest or abdominal trauma diverts the attention

of the examining physician. This oversight is less apt to occur when there is only a single injury, such as a direct blow to the neck area. Even in this situation, however, it should be remembered that laryngeal edema, ecchymosis or emphysema may develop some hours after injury and the resulting airway obstruction may create an emergency which might have been avoided had the possibility of such a crisis been anticipated. The diagnosis of laryngeal injury begins with the examiner who suspects its possibility. Examination by palpation, mirror, radiographic studies and direct laryngoscopy is of basic importance. If there are any signs of airway obstruction, tracheostomy should be done early as an elective procedure rather than later as a desperate life-saving measure. In some instances of severe crushing injuries, provision of an airway becomes an immediate and prime necessity.

It is particularly important, however, that definitive therapy be instituted early in order to prevent the scarring and stenosis which will take place if the contour of the larynx is not maintained during the healing period.

The laryngologist will be required to choose between an external or internal approach for splinting the acutely injured larynx.[6] Debridement of fragments, control of bleeding and introduction of a splint through a thyrotomy incision may be necessary in some cases. In others, a direct laryngoscopic approach may be used to reduce dislocations of cartilage. A rubber, polyethylene or acrylic splint may then be introduced and anchored to the tracheostomy tube or the neck by strings. These measures are adopted for acute injuries. If stenosis of the larynx should occur as a late complication of a neglected laryngeal injury, restoration of the lumen by scar tissue excision, skin grafting and splinting will ultimately become necessary.

Illustrative Case

Case V. A 41 year old housewife was thrown against the dashboard, steering wheel and front window of her car when she struck another automobile on an expressway. When seen in the emergency room, an extensive jagged scalp laceration which exposed the vertex of her skull seemed to attract the most attention. The neurological service found no evidence of severe head injury but during this examination it became evident that hoarseness, dyspnea and stridor were developing fairly rapidly. Subcutaneous air in the neck was noted. Mirror examination revealed edema, submucosal blood and distortion of the intralaryngeal structures.

An elective tracheostomy was performed before the patient's airway problem became acute. Blood and air bubbles were encountered in the fascial planes around the strap muscles. A tracheal cannula was inserted between the third and fourth tracheal rings. Immediate relief of dyspnea was evident. Following this, extensive debridement and suturing of the scalp laceration was carried out.

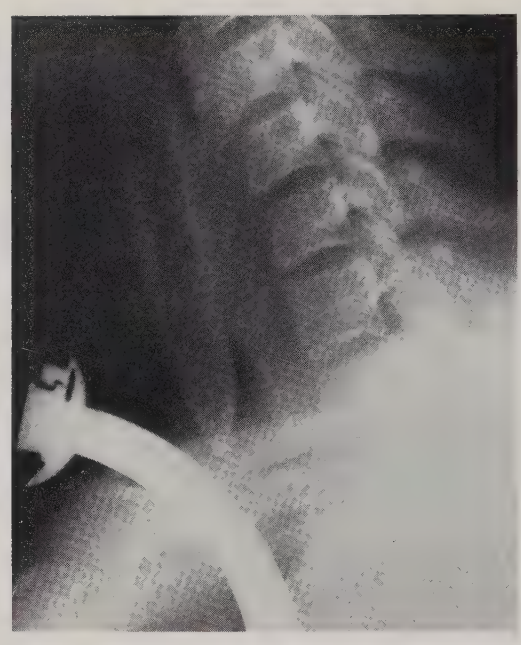

Figure 6 (Case V). Roentgenogram showing tracheal cannula in position with a polyethylene stent superior to it as a splint in the larynx. Frequent changes and gradually increasing sizes of such stents are used in the prolonged after-care of laryngeal injuries until permanent healing and epithelialization are established.

Four days later a direct laryngoscopy was performed under general anesthesia. Several fragments of sequestrated cartilage were removed from the larynx. The right vocal cord was fixed but motion remained in the left cord. A polyethylene mold was inserted into the larynx and secured by strings brought out through the tracheostomy opening and tied around the neck (Fig. 6).

During the ensuing 5-month period there were numerous changes of polyethylene splints of gradually-increasing sizes. Granulation tissue was removed on several occasions. The larynx gradually epithelialized and the normal anatomy became re-established. The right cord remained fixed but complete return of motion of the left cord permitted a normal exchange of air and resulted in a good voice. The tracheal cannula was finally removed after 6 months and the patient returned to her secretarial position.

Comment. This particular clinical history represents a fairly typical case. The patient was fortunate in that definitive treatment could be initiated at an early stage. The choice of which method to pursue in treating this type of injury is dependent upon a careful evaluation of the extent of the laryngeal disruption. A combination of methods may

be necessary to achieve a successful final result. Continued follow-up observation of these patients is necessary since late scar tissue contraction and narrowing of the airway may occur following an apparent initially satisfactory outcome.

SUMMARY

Glottic rehabilitation in vocal cord paralysis by means of Teflon injection, conservation of laryngeal function by subtotal laryngectomy, combined radiation and surgery for extensive cancer of the larynx and the management of laryngeal trauma have been described and illustrated by case reports.

REFERENCES

1. Arnold, G. E.: Vocal rehabilitation of paralytic dysphonia. Arch. Otolaryng. *76:* 352–368 (Oct.) 1962.
2. Clinical Staging System for Cancer of the Larynx: Joint Committee on Cancer Staging and End Results Reporting, 40 East Erie St., Chicago, Ill. 60611, 1962.
3. Friedberg, S. A. and Wallner, L. J.: Selected problems in the diagnosis of laryngeal carcinoma. A.M.A. Arch. Otolaryng. *58:* 521–535 (Nov.) 1953.
4. Goldman, J. L. et al.: Combined radiation and surgical therapy for cancer of the larynx and laryngopharynx. Laryngoscope *74:* 1111–1134 (Aug.) 1964.
5. Hendrickson, F. R.: Personal communication.
6. Henry, G. A.: Laryngeal trauma. Canad. M. A. J. *88:* 833–836 (April) 1963.
7. Leroux, R. J.: Indications for radical surgery, partial surgery, radiotherapy and combined surgery for cancer of the larynx and hypopharynx. Ann. Otol., Rhinol. & Laryng. *65:* 137–153 (March) 1956.
8. Lewy, R. B.: Glottic reformation with voice rehabilitation in vocal cord paralysis. Laryngoscope *73:* 547–555 (May) 1963.
9. Ogura, J. H.: Surgical pathology of cancer of the larynx. Laryngoscope *65:* 867–926 (Oct.) 1955.
10. Ogura, J. H.: Supraglottic subtotal laryngectomy and radical neck dissection for carcinoma of the epiglottis. Laryngoscope *68:* 983–1003 (June) 1958.

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Microsurgery of the Ear

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The advances that have been made in surgery of the ear during the past three decades have been rapid and in some instances even dramatic. While accumulation of basic information has been fairly continuous, there have been several outstanding contributions in the development of surgical techniques.

This report will be confined to a discussion of three procedures, all of which entail the use of the operating microscope, hence classed under "microsurgery." These are stapes surgery for improvement of hearing in otosclerosis, surgery for prevention of episodes of vertigo in Meniere's disease, and the translabyrinthine surgical approach to the internal auditory meatus for removal of small acoustic neuromas.

As a preface to discussion of these procedures, a review of certain factors which have contributed to the evolution of modern ear surgery is necessary.

A detailed knowledge of anatomy and pathology of the temporal bone with its contained sense organs has been essential. Microscopic studies by means of serial sections of the temporal bone and reconstructions have provided much basic detailed information about the histopathology of otosclerosis, Meniere's disease, early neuromas in the internal auditory meatus, and petrositis. These, and also the studies of physiology of the ear, utilizing recorded electrical potentials and behavioral techniques to determine function in the animal, have provided background for development of modern surgical procedures.

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Figure 1. Otosclerosis. Photomicrograph of stapes footplate and one crus. The stapes footplate is many times its normal thickness. Otosclerotic bone involves the margins of the oval window, the footplate and the base of the crus. (Lindsay, J. R.: Histopathologic changes following fenestration and stapes mobilization. Tr. Am. Acad. Ophth. & Otol. *63:* 187–205 [March-April], 1959.)

SURGERY FOR DEAFNESS DUE TO OTOSCLEROSIS

Otosclerosis is a disease of bone limited to the bony capsule of the labyrinth (Fig. 1). It occurs usually in limited foci, most often in front of the oval window but sometimes also at the round window and at the inner end of the internal meatus. It causes hearing impairment by extension into and ankylosis of the stapes in the oval window. Some degree of sensorineural impairment frequently follows. The disease is most common in Caucasians, infrequent in the Negro and only occasional in Chinese and Japanese. There is a hereditary factor and if hearing impairment appears it is usually in the second and third decades. The cause is unknown and no medical therapy has been effective to date.

Surgical attempts to restore function were being made in the eighties and nineties of the last century. The approach was through the ear drum and attempts were made to mobilize the fixed stapes or to remove it. Some success was achieved temporarily, particularly by French otologists, but the procedure was discontinued as unsatisfactory.

The Fenestration Operation

Attempts to bypass the fixed stapes and create a new window at some other location in the wall of the bony labyrinthine capsule, to permit the entry of sound, were continued on the part of a few pioneers. The work of Holmgren[7] of Sweden stands out, and on the basis of that work Sourdille[20] of France successfully developed a two-stage procedure to make a permanent window into the horizontal semicircular canal covered by thin skin in the early 1930's.

During this period the major problem in surgery of the ear was that of bacterial suppuration in the deeper parts of the petrous pyramid leading to fatal intracranial complications.

Skills achieved in surgery of the temporal bone in dealing with petrositis did much to prepare the way for the introduction by Lempert[11] of New York of his one-stage fenestration operation in 1938 (see Fig. 2). The contribution of this surgical technique and the refinement in all surgery of the temporal bone that followed Lempert's teaching mark an outstanding advance in otologic surgery.

Although this procedure was capable of restoring hearing to a practical level without a hearing aid in selected cases, there were certain

Figure 2. Fenestration operation. Photomicrograph showing the fenestra (arrows) in the wall of the bony semicircular canal covered by a thin layer of skin obtained as a flap from the posterior and superior external canal wall. Hearing was restored to the practical level. (Lindsay, J. R.: Histopathologic changes following fenestration and stapes mobilization. Tr. Am. Acad. Ophth. & Otol. *63:* 187–205 [March-April], 1959.)

deficiencies. The loss of the action of the ear drum and ossicular chain limited the improvement theoretically possible by about 25 decibels in all cases. Hence, only those patients with relatively good inner ear function could be expected to have restoration of unaided hearing to a practical level. Since a large percentage of otosclerotics are found to have some degree of sense organ (sensorineural) impairment progressing with age, many could not obtain sufficient improvement by this procedure.

The fenestration operation also creates a radical mastoid cavity lined by skin which has a disadvantage in that periodic cleaning is usually necessary to prevent secondary infection in the cavity.

Stapes Surgery

The rediscovery by Rosen[17] that the fixed stapes could sometimes be mobilized successfully created immediate interest. If the stapes would remain mobile, it would mean the possibility of up to 25-decibel improvement in hearing over that obtainable with the fenestration procedure. The external canal and ear drum would remain intact. It would mean that the method could be used advantageously in the presence of moderate degrees of secondary sensorineural impairment.

Two other developments since Lempert introduced his fenestration operation have made the development of modern microsurgery of the ear possible. These are the greater security from bacterial infection provided by antibiotics and, of more importance technically, the perfection of the modern operation microscope, the Zeiss otoscope, which permits magnification from ×6 to ×40, with a working distance of up to 300 mm. The operating microscope permitted the development of the meticulous techniques essential to all types of surgery on the sound conduction apparatus, the inner ear and the internal auditory meatus.

A disadvantage in the development of stapes surgery has been the fact that the disease has never been found in animals. The technique has therefore been developed almost entirely in the human and its refinement has been slow since the risk of injuring the inner ear had to be assessed.

MOBILIZATION OF THE STAPES. The method used by Rosen[17] to expose the middle ear consisted in elevating a cuff of posterior bony canal wall skin down to the drum, elevating it with its fibrous attachment and displacing it forward. The method was already in use for other purposes and has become standard. After discovering that a fixed stapes might be loosened, Rosen devised the method of exerting pressure on the neck of the stapes, directed toward the posterior crus. The stapes was either mobilized with sudden improvement in hearing or the stapes was fractured (see Fig. 3). The intact drum was then replaced.

Although there was skepticism, later justified, about the permanency

of the mobility thereby achieved, the potential advantages to be gained over the standard fenestration procedure were apparent.

The procedure was popular with the patient because of its simplicity. It appealed to otologists untrained in fenestration surgery as a technique mistakenly thought to be easily mastered. It was evident within a very few years that a relatively small percentage of cases maintained the hearing level achieved by mobilization and that a high percentage of stapes could not be mobilized by simple manipulation. The reasons for failure were apparent both on observation with the operating microscope and microscopic examination in the laboratory.

Modification of the Mobilization. During the next few years a succession of procedures were developed by a number of surgeons, all of which were aided by the use of the microscope. These progressed from attempts to mobilize the footplate directly by the use of picks and fine chisels to a variety of methods based on fracturing the footplate on the erroneous theory that fractures of the footplate healed only by fibrous union.

Some of these procedures proved suitable in carefully selected cases.

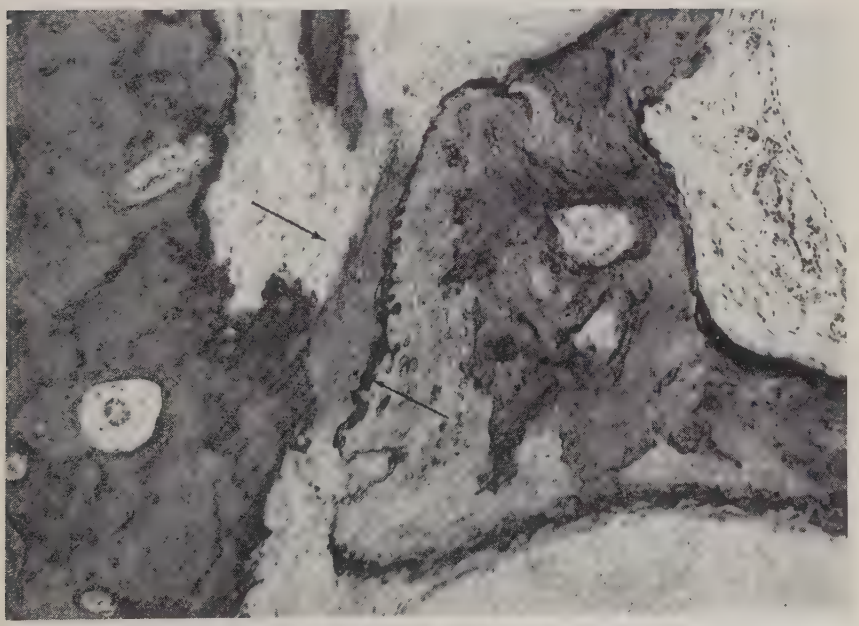

Figure 3. Mobilization of the stapes. High power photomicrograph to show ankylosis by new bone (arrows) which is forming in the annular ligament between the footplate of the stapes and the rim of the window one year after operation. The rim of the window is composed of otosclerotic bone which replaced the normal endochondral bone of the labyrinthine capsule.

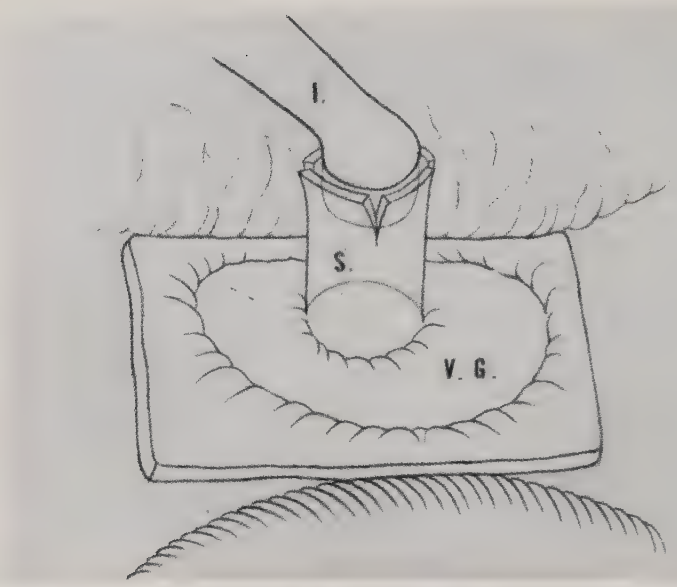

Figure 4. Total stapedectomy. Drawing to illustrate a vein graft (*V. G.*) covering the oval window and connected to the incus (*I.*) by a polyethylene tube strut (*S.*). (Courtesy of John J. Shea, Jr.: Fenestration of the oval window. Ann. Otol., Rhin. & Laryng. *67:* 932–951, 1958.)

However, the incidence of refixation was high. Fractures of the footplate tended to reunite, particularly if thickened by otosclerosis.

TOTAL STAPEDECTOMY. The discovery that the stapes could safely be removed completely and a graft used to cover the oval window led to two significant developments in stapes surgery.

1. Shea[19] of Memphis developed the technique of applying a small graft consisting of the wall of a vein from the hand over the oval window after removal of the stapes (Fig. 4). A piece of fine polyethylene tubing about 4 mm. in length was inserted, one end beveled to help maintain its position on the vein graft and the other fitted over the tip of the long process of the incus.

2. Schuknecht[18] developed the technique of removing the stapes and using a piece of extremely fine steel or Tantalum wire of which one end was tied around a tiny mass of subcutaneous connective tissue or a lump of fat removed from the patient's ear lobe and used as a graft in the oval window, the other end then being crimped around the long process of the incus.

Both methods were used extensively for several years and were reported successful in restoring the conduction mechanism to a nearly normal state in over 80 per cent of cases, depending in part on the type of case selected for surgery.

Some modifications of these methods also appeared, such as a combination of wire and vein, and gave similar results.

Failures and Complications. Failures occurred for several reasons, including mainly technical difficulties, unusual features of the disease, a noninfectious labyrinthine reaction to the graft, and hemorrhage from the vascular otosclerotic bone into the vestibule.

Profound loss of function occurred in a few patients owing to postoperative labyrinthine reaction. Late complications occurred in a small percentage. The causes, although sometimes unexplained, included perforation of the strut into the inner ear following extreme change in air pressures, and invasion of the inner ear from a bacterial or viral middle ear infection.

Current Methods of Stapedectomy. In the past two years the use of a polyethylene tube strut has been largely supplanted by steel or tantalum wire as less susceptible to complications since the wire retains its attachment to the incus during any extreme movement. The wire is well tolerated and usually becomes covered by an extension of the middle ear epithelium.

Vein, fat and connective tissue have been suspected of causing an irritative reaction in the inner ear and have therefore lost favor. Two methods currently popular in America are the use of a small pad of gelfoam placed in the oval window and connected to the incus by wire (Fig.

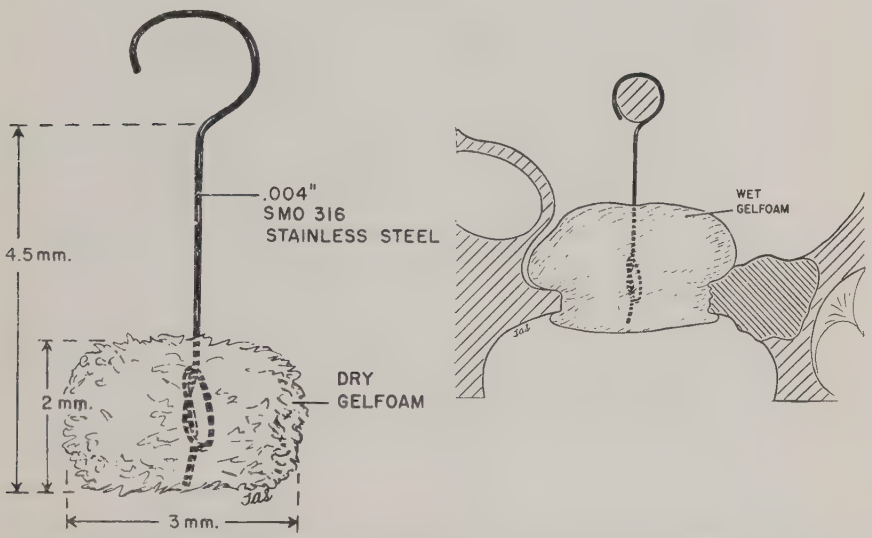

Figure 5. Total stapedectomy. Diagrams to illustrate the gelfoam pad placed in the oval window and attached by stainless steel wire to the long process of the incus. (Courtesy of Harold F. Schuknecht: A new stapedectomy prosthesis. Arch. Otolaryng. *80:* 474, 1964.)

5), and also the use of a tiny cylindrical or oval piston of Teflon or steel attached to the incus by wire and extending just through the oval window into the vestibule (Fig. 6). The solid piston has been favored in cases in which the otosclerosis in the oval window niche has been extremely thick, as a means more likely to prevent refixation by regrowth of bone.

Gelfoam and wire has been favored because of its lack of irritation of the inner ear and tendency to absorb, leaving a thinner membrane over the window than in the case of tissue grafts. The thin membrane may provide less impedance to sound transmission. Late complications have occurred with both methods. The efficiency of the wire and gelfoam method in restoring conduction has been high.

Some question remains as to the ability of either device to protect the inner ear from extension of a bacterial or viral middle ear infection as efficiently as the normal stapes. However, the incidence of these infections in the adult is very low and late complications reported to date have been few. Experience over the past five years has provided increasing confidence in the permanency of the new oval window.

The sensorineural component of the deafness is not impeded in its progress by the surgical restoration of hearing and may produce a serious handicap in a considerable number in the advanced stages of the disease.

Modern hearing aids function well in overcoming the conduction impairment due to otosclerosis. Although the hearing function as restored by surgery is preferred by the patient, the electrical hearing aid is essential in certain circumstances, i.e., to help overcome a remaining sensorineural deficit after surgery; also when the patient is wholly dependent on one ear.

The ultimate answer to the otosclerosis problem must be either in its prevention or in the development of a method of arresting its progress.

SURGERY FOR THE RELIEF OF DIZZY SPELLS IN MENIERE'S DISEASE

The clinical syndrome now known as Meniere's disease was first described by Meniere[14] over 100 years ago. Since 1938 when Hallpike and Cairns[6] demonstrated an idiopathic type of "hydrops" or dilatation of the endolymphatic fluid system as the pathology in the inner ear the name "Meniere's disease" has been widely adopted.

Symptomatology

The disease involves both the cochlear and vestibular labyrinths. Auditory symptoms and findings are characteristic and provide the diagnosis. They consist of a fluctuating hearing impairment, mainly for low tones by air and bone conduction, along with low- and medium-pitched

Figure 6. Photomicrograph of a section of a polyethylene tube strut which had been inserted over a vein graft in the oval window and fitted onto the incus above. The window has been narrowed by the formation of new bone between the vascular otosclerotic bone and the tip of the strut. The window has been thereby narrowed but not completely closed off. Elastic tissue in the vein wall on either side of the strut is stained black.

ear noises, sometimes diplacusis and lowered tolerance for loud sounds. Recruitment of loudness occurs with increasing intensity of the sound until very loud sounds may be louder in the impaired ear than in the normal. Distortion is thereby produced.

Dizziness occurs in episodes, with rapid or sudden onset and usually rapid cessation. Duration and severity vary. The onset may be without warning and the frequency of attacks varies widely.

The condition is bilateral in about 10 per cent of cases.

As the deafness advances, the ability to discriminate speech decreases so that amplification by means of a hearing aid is of limited value.

While the auditory symptoms may be distressing and a severe handicap if bilateral, the vertigo may be incapacitating and hazardous because of the sudden onset, sometimes with falling. The patient usually seeks help to get relief from these recurring episodes.

The cause of the disease is not known nor is the pathogenesis of the "hydrops" explained.

Histopathology

Hydrops of the cochlear duct has been related to the auditory disturbances and these usually precede the appearance of dizzy spells.

Dilatation of the scala media (cochlear duct) is a constant finding (Fig. 7). The neural elements and the hair cells in Corti's organ are usually not decreased in number as compared to the normal ear. In the majority of cases the saccule is also dilated (Fig. 8) and may occupy all of the space in the vestibule normally occupied by perilymph.

In the occasional case the dilated cochlear duct (Reissner's membrane) may fill the available space in the vestibule. The vestibular part of the labyrinth also shows distortions and possible ruptures of the membranes at the junction of the ampullae of the semicircular canals and the utricle (Figs. 8 and 9). The spells of vertigo are probably related to these localized distortions and occur on a physical basis as temporary disturbance of function at an ampulla.

Medical Therapy

Clinical observation shows that the disease fluctuates widely. Medical therapy has been varied and based on a variety of theories as to etiology. The normal fluctuations and remissions of the disease complicate the evaluation of therapy. Factors such as stress and nervous fatigue seem to increase the symptoms.

Surgical Procedures

Surgical intervention is used primarily to prevent episodes of vertigo. Two general types of surgery have been employed—one to prevent vertigo

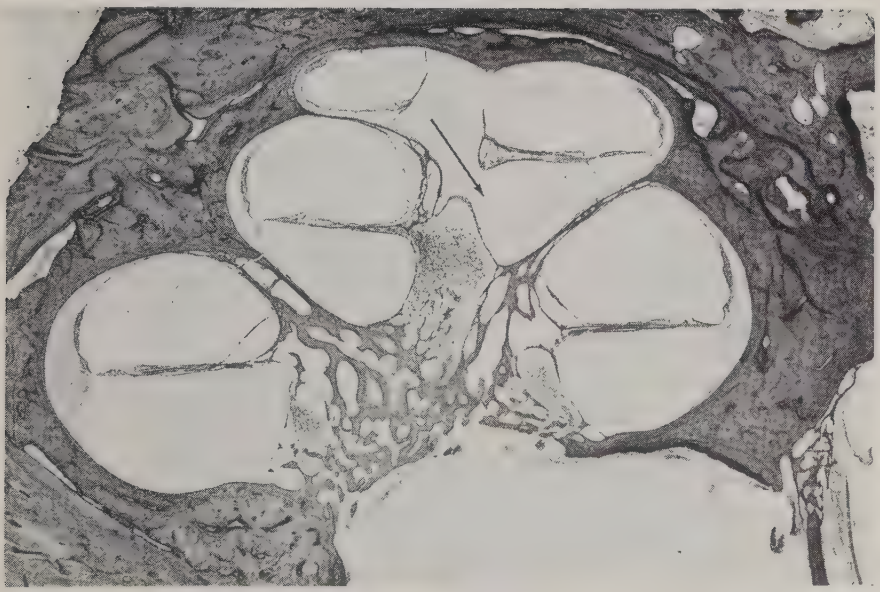

Figure 7. Photomicrograph of midmodiolar section of the cochlea in Meniere's disease. The scala media (cochlear duct) which contains the sensory receptors is dilated to occupy almost all of the vestibular scala and has extended through the helicotrema into the scala tympanum at the apex (arrow).

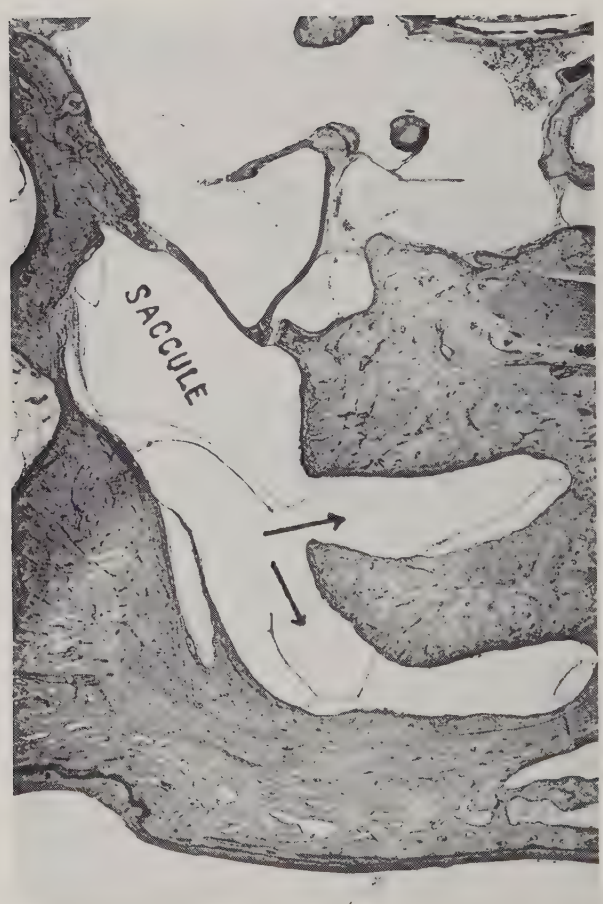

Figure 8. Photomicrograph of the vestibule of an ear with Meniere's disease showing the greatly dilated saccule which is in contact with the footplate of the stapes, and two herniations from the utricle (arrows) extending into the perilymphatic space of the small end of the horizontal canal and the common crus of the vertical canals.

Figure 9. Photomicrograph of a vertical section through the ampullae of the superior, horizontal and posterior semicircular canals showing a portion of the macula of the utricle. Herniations or distortions which have occurred at the weak points in the utricular walls are indicated by arrows. These localized weakened areas are located at the junction of the various canals with the utricle.

while preserving hearing and one to destroy both the vestibular and auditory sensory apparatus.

LABYRINTHECTOMY, OR TOTAL DESTRUCTION OF INNER EAR FUNCTION. Total destruction of inner ear function is the preferred method when the hearing threshold and the ability to discriminate sounds in that ear are of little or no value. It offers the greatest assurance of preventing episodes of vertigo and is followed by some reduction of the tinnitus.

Several methods of destroying labyrinthine function have been used. Intracranial section of the eighth nerve was used by Dandy[3] of Baltimore and abandoned in favor of intracranial division of the vestibular part of the eighth nerve, to avoid destruction of hearing, as first proposed by McKenzie[13] and Ireland of Toronto.

Labyrinthotomy and ablation of the membranous horizontal semicircular canal[2] and coagulation of the canal ampulla[4] were widely used, both with relatively satisfactory results, but with an occasional recurrence of attacks.

Labyrinthectomy following mastoidectomy. Exenteration of the horizontal and anterior vertical canal ampullae and the utricle following mastoidectomy was used and preferred by the authors as a more certain means of destroying the function and assuring the prevention of further attacks of dizziness.

Labyrinthectomy by the middle ear approach (Fig. 10), under local anesthesia with procaine or zylol and epinephrine, is at present the method favored by the authors. The procedure is carried out under the microscope.

Under suitable premedication and preparation of the ear the middle ear is exposed by incising the skin of the bony canal wall about 5 mm. external and parallel to the posterior margin of the drum. Elevation of the skin, periosteum and posterior half of the drum and removal of the posterior inner margin of the bony wall permits a full view of the incudostapedial joint, the oval and round windows and the promontory of the inner ear. The patient is then given an intravenous injection of meperidine (Demerol), 50 to 75 mg., before the vestibule is opened.

The stapes is then taken out and the bony promontory removed by use of the electric burr down to the round window, exposing the contents of the vestibule. The utricle with attached canals and the saccule are then removed. The vestibule is packed with gelfoam and the drum replaced. One small strip of gelfoam is placed over the incision, supported by a small strip of chlortetracycline (Aureomycin) impregnated gauze.

The sedation is usually adequate, bleeding minimal and visualization good. Removal of the vestibular contents is tolerated well. Antibiotics are given in the postoperative period.

Total destruction of one labyrinth produces vertigo and disequilibrium which subsides gradually in the following weeks. Some persistence of

disequilibrium on quick movements should be anticipated for many months, until compensation is well developed.

Selective and Partial Destruction of Vestibular Function. *Intracranial Vestibular Nerve Section.* This procedure has usually been carried out by the neurosurgeon by the posterior fossa approach.

In recent years a surgical approach to the internal auditory meatus by way of the middle fossa has been described and used by William House[8] of Los Angeles, using the microscope and electrically driven burrs. The method provides a view of structures in the meatus and permits sectioning of the vestibular nerve, or removal of a very small tumor. Both methods involve opening the subarachnoid space.

Operations on the Endolymphatic Sac.

drainage of the sac. Portmann[15] of Bordeaux devised an operation for drainage of endolymph from the endolymphatic sac to relieve supposed pressure many years before a hydrops of the endolymphatic spaces was known to exist. The Portmanns[16] continue to report good results in preventing the episodes of vertigo. By experiments on the monkey, however, it was demonstrated in this clinic[12] that surgical open-

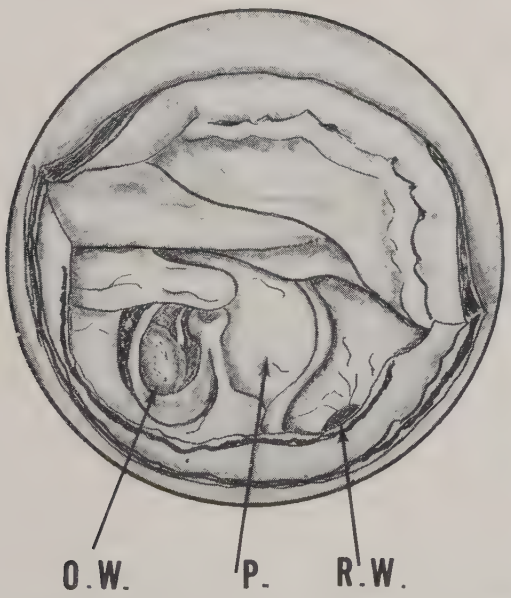

Figure 10. Labyrinthectomy by middle ear approach. A drawing to show the exposure of the middle ear obtained by reflection of a flap of skin and periosteum from the posterior canal wall down to the drum. The flap and drum have then been reflected forward and bone removed sufficiently to expose the promontory (*P.*), the oval window niche containing the stapes (*O.W.*), and the round window niche (*R.W.*). In the operation the promontory is removed thereby connecting the two windows and exposing the contents of the vestibule.

ing or destruction of the endolymphatic sac is quickly healed and new bone deposited in the area.

THE SHUNT OPERATION. In the past few years an operation was developed by William House[9] to expose the endolymphatic sac, which projects into the dura from a niche in the temporal bone posterolateral to the meatus, and create a shunt between the sac and the subarachnoid space by insertion of a small plastic tube or button. The method is based on the assumption that the pressure within the endolymphatic spaces will thereby be stabilized and disturbances of function avoided. Experience with this operation is at present inadequate to permit its evaluation.

Surgical Rupture of the Saccule. Studies of the histopathology have shown that in Meniere's disease the saccule usually is dilated, with its wall in contact with the footplate of the stapes. In some cases, however, the dilated cochlear duct may bring Reissner's membrane in contact with the stapes. A microsurgical operation has been devised by Fick[5] of South Africa consisting of rupturing the saccule through a hole made into the vestibule at the lower margin of the stapes footplate. This procedure requires further clinical trial and experimental investigation for evaluation.

Ultrasonic Therapy for Meniere's Disease

Selective destruction of function of one or more semicircular canals by ultrasound has been under development for several years. Favorable results by Arslan[1] of Padua in preventing vertigo in Meniere's disease led to further improvements in equipment and technique in several centers, notably in Uppsala by Sjoberg and associates and in England by Angell James. The objective is to prevent episodes of vertigo and avoid further loss of hearing.

Under local anesthesia the mastoid cells are exenterated. The mound of the horizontal canal is next exposed and under microscopic control it is flattened by means of suitable burrs until the lumen of the canal can be distinguished. The covering bone is left intact.

The tip of the ultrasound applicator is placed over the prepared area and treated by the ultrasonic beam of measured intensity. The point of contact is kept cool by a drip of saline and suction. Eye movements and facial function are observed constantly. Treatment is continued until nystagmus is constantly directed to the opposite ear, indicating canal paralysis, or for a total of 20 minutes. Moderate dizziness on movement of the head is usually experienced following treatment and for a few weeks thereafter.

The destruction of function obtained by the treatment of one canal has been found satisfactory in about 70 per cent and hearing preserved in over 80 per cent. A second treatment may be given if necessary.

SURGERY FOR ACOUSTIC NEUROMA

Dr. William House of the Los Angeles Otological Medical Group has revitalized interest in the translabyrinthine approach to the acoustic neuroma.[10] By means of the operating microscope and the electrically driven drill he has been able to perform an operation which was once considered too difficult and too dangerous to be practical. The main advantages in this approach lie in that it allows for direct visualization of the facial nerve during the removal of the tumor and produces a minimum of trauma to the adjacent structures such as the brain stem and cerebellum. It seems to be best suited for the total removal of the small early tumor.

Diagnosis

Since these tumors arise from the eighth cranial nerve, the earliest symptoms will be due to alteration of function of that nerve. Only as the tumor enlarges will it cause alterations of functions of adjacent structures such as the facial nerve, the trigeminal nerve and cerebellum. Of utmost importance is an increase in the index of suspicion in the examining physician who first sees the patient with a unilateral hearing loss or vestibular complaints.

Diagnostic tests should include complete audiologic and vestibular evaluation, radiographic studies (including polytomography, if available, and contrast studies) and a complete neurologic examination.

Audiometric Testing. Modern audiology now has at its disposal several tests that are useful in localizing the lesion responsible for producing hearing loss. In the search for a retrocochlear loss as would be produced by an acoustic neuroma, a battery of tests is required. These should include pure tone audiograms by air and bone conduction, auditory discrimination using standard PB word tests, alternate binaural loudness balance (ABLB), the short-increment sensitivity index (SISI), Bekesy audiometry and tests for tone decay. Classically, the acoustic neuroma will demonstrate a sensorineural hearing loss with discrimination scores that may be disproportionately low. The ABLB test will usually not show recruitment and the SISI will be negative. A positive response to these tests is an indication of cochlear dysfunction such as is found in Meniere's disease. Tone decay is a phenomenon of retrocochlear lesions and is also demonstrated by Bekesy audiometry, which gives a Type III or IV tracing.

Vestibular Testing. The vestibular portion of the eighth nerve is usually affected early, therefore vestibular function must also be tested. This may be done by rotation tests or by caloric stimulation. The latter is preferred since it permits the ears to be stimulated separately. A variety

of methods are in use but most suitable are the Kobrak minimal stimulation test and the method described by Hallpike of using hot and cold stimulation alternately. Patients with an early acoustic neuroma will usually show some decrease and later absent vestibular response on the involved side. Vestibular paresis or paralysis from other causes is also not uncommon and there is no qualitative method of distinguishing that of the acoustic neuroma. Electronystagmography generally does not yield any further diagnostic information, but it does serve as a useful permanent record of the patient's nystagmus and permits a more quantitive estimation of altered function.

RADIOLOGIC EXAMINATION. Radiographic studies of the petrous pyramids may show asymmetry or enlargement of the internal auditory meatus due to the presence of tumor. The most useful views are the Cauldwell, Towne-Chamberlain and Stenvers. Recently the addition of polytomography and pantopaque studies has provided a definite aid in distinguishing small tumors that may still be located within the meatus. Pantopaque contrast studies may also yield additional information concerning adjacent blood vessels.

NEUROLOGICAL EXAMINATION. The neurological examination should pay particular attention to the fifth and seventh cranial nerves. The House group has given particular attention to the quantitative evaluation of trigeminal and facial nerve function. Their studies have indicated that these nerves may be involved early in the course of development of the acoustic neuroma. Ordinarily acoustic tumors do not cause trigeminal nerve signs until there is actual mechanical pressure on a nerve trunk within the cerebellopontine angle. Therefore, the earlier the tumor is diagnosed the fewer will be the trigeminal signs. Tests of trigeminal nerve function should attempt to detect an alteration in corneal sensitivity, the pain threshold of the face, and a difference in the ability to use the masseter muscles. Usually, corneal sensitivity will be first altered. Hypesthesia may be an early symptom.

The facial nerve may be subjected to the pressure within the internal auditory meatus by the enlarging tumor. The House group has measured the blink reflex with special high speed photographic equipment to evaluate facial nerve motor function, a definite delay being noted in a significant number of patients on the side involved with the tumor. Sensory functions of the facial nerve may be tested by stimulating the anterior one-third of the tongue either electrically or chemically. The parasympathetic function of the facial nerve is assessed by measuring the production of tears by placing filter paper strips into the conjunctival sacs.

CEREBROSPINAL FLUID. The cerebrospinal fluid protein is classically elevated with the acoustic neuroma. However, with the early tumor it may be entirely normal. It must also be remembered that other

diseases such as ruptured intervertebral disk, cord tumor or multiple sclerosis may also elevate the cerebral spinal fluid protein.

Differential Diagnosis and Treatment

The early acoustic neuroma must be differentiated from Meniere's disease and deafness of sudden onset due to viral or vascular disease. The course to be taken when the presence of tumor is still suspected after all tests including pantopaque studies have been negative will depend mainly upon the degree and progress of sensorineural hearing loss in the involved ear.

If the hearing is of a degree which is worth preserving, a middle fossa approach as described by House should be considered. If a small tumor is found it may be removed. This approach also permits the sectioning of the vestibular nerve for relief of vertigo, if indicated.

If the hearing is no longer useful and the tumor small, a translabyrinthine approach should be considered. This approach will destroy both vestibular and auditory function. House has demonstrated that the small tumor may be removed with relative ease by this method with direct visualization of the facial nerve and, therefore, less chance of injury to it.

The prevailing opinion seems to be that for the large tumor the standard suboccipital approach is probably the safest and most effective procedure and may permit total removal. The facial nerve is vulnerable to injury in that the tumor must be removed before the nerve can be seen; however, a nerve anastomosis or a graft can be used to restore at least partial function.

All of these surgical procedures require considerable skill, judgment and cooperation between the otolaryngologist and neurosurgeon. It is through such a team approach concept that we can offer the patient with the acoustic neuroma the best possible treatment for the disease. It is clear also that the role of the otolaryngologist is changing from one of a diagnostician to one who can actively participate in the treatment of this disease.

REFERENCES

1. Arslan, M.: An improved technique of the ultrasonic irradiation of the vestibular apparatus by Meniere's disease. Acta otolaryng. *55:* 467–472, 1962.
2. Cawthorne, T. E.: Treatment of Meniere's disease. J. Laryng. & Otol. *58:* 363, 1943.
3. Dandy, W. E.: Surgical treatment of Meniere's disease. Surg. Gynec. & Obst. *72:* 421–425 (Feb.) 1941.
4. Day, K. M.: Hydrops of labyrinth (Meniere's disease). Diagnosis—Results of labyrinth surgery. Laryngoscope *56:* 33–42 (Feb.) 1946.

5. Fick, I. A. van.: Decompression of the labyrinth. A new surgical procedure for Meniere's disease. Arch. Otolaryng. *79:* 447–458 (May) 1964.
6. Hallpike, C. S. and Cairns, H.: Observations on the pathology of Meniere's syndrome. Proc. Roy. Soc. Med. *31:* 1317–1336, 1938.
7. Holmgren, Gunnar: Surgery of otosclerosis. Ann. Otol., Rhin. & Laryng. *46:* 3–12 (March) 1937.
8. House, William F.: Surgery of the Petrous Portion of the VII Nerve. Ann. Otol., Rhin. & Laryng. *72:* 802-807, 1963.
9. House, W. F.: Subarachnoid shunt for drainage of endolymphatic hydrops. Laryngoscope *72:* 713–729, 1962.
10. House, W. F. (Ed.): Monograph. Transtemporal bone microsurgical removal of acoustic neuromas. Arch. Otolaryng. *80:* 601–756 (Dec.) 1964.
11. Lempert, Julius: Improvement of hearing in cases of otosclerosis. A new, one stage surgical technic. Arch. Otolaryng. *28:* 42–97 (July) 1938.
12. Lindsay, J. R.: Effect of obliteration of the endolymphatic sac and duct in the monkey. Arch. Otolaryng. *45:* 1–13 (Jan.) 1947.
13. McKenzie, K. G.: Intracranial division of the vestibular portion of the auditory nerve for Meniere's disease. Canad. M.A.J. *34:* 369–381 (April) 1936.
14. Meniere, P.: Memoire sur des lésions de l'oreille interne donnant lieu à des symptomes de congestion cérébrale apoplectiforme. Gaz. méd. de Paris, s 3, *16:* 597–601, 1861.
15. Portmann, G.: The saccus endolymphaticus and an operation for draining the same for the relief of vertigo. J. Laryng. & Otol. *42:* 809–817 (Dec.) 1927.
16. Portmann, Michel: Decompressive opening of endolymphatic sac. Arch. Otolaryng. *79:* 328–337 (April) 1964.
17. Rosen, S.: Mobilization of the stapes to restore hearing in otosclerosis. New York J. Med. *53:* 22 (Nov. 15) 1953.
18. Schuknecht, H. F.: A new stapedectomy prosthesis. Arch. Otolaryng. *80:* 474–475 (Oct.) 1964.
19. Shea, J. J., Jr.: Fenestration of the oval window. Ann. Otol., Rhin. & Laryng. *67:* 932–951, 1958.
20. Sourdille, Maurice: Sur le traitement chirurgical de l'otosclerose. Rev. laryng. *51:* 341–353 (May 30) 1930.

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A New Technique Employing Partial Temporal Bone Resection in Parotid Gland Surgery for Cancer

GEORGE W. ALLEN, M.D.*

Although most surgeons dealing with malignant tumors in the region of the ear will remove portions of the mastoid and ear canal when it becomes necessary, only recently have attempts been made to systematize these maneuvers. In the operation to be described here, a partial temporal bone resection is the new feature that has been added to the standard form of total parotidectomy and neck dissection. This is one aspect of a rapidly developing field that has been termed "surgery of the base of the skull." This field includes transnasal or trans-sinus hypophysectomy, translabyrinthine removal of acoustic nerve tumors, decompression of the eighth cranial nerve medial to the geniculate ganglion by a middle fossa approach, and endolymphatic sac decompression for Meniere's disease.

The rationale for this operation is based on the fact that there are prolongations of the parotid gland into anatomical recesses that interfere with its clean, complete removal. Two prolongations extend on either side of the styloid process, one into the petrotympanic fissure which lies in front of the bony external ear canal and behind the condyle of the mandible, and the other into the tympanomastoid fissure which runs laterally from the stylomastoid foramen containing the facial nerve (Fig. 1). It was decided that for tumors involving these areas, the resection should include a margin of uninvolved bone in order to insure complete removal.

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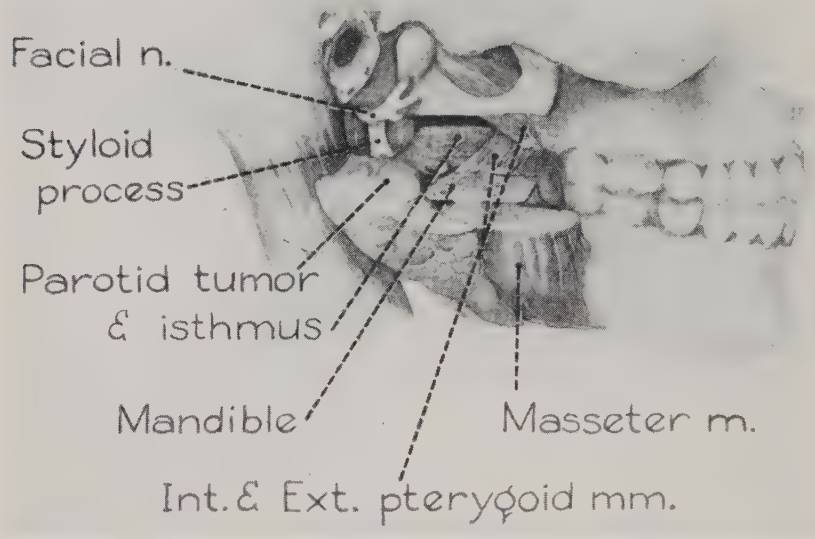

Figure 1. The critical regions where removal of carcinoma of the parotid gland is most likely to be inadequate lie near the exit of the facial nerve from the stylomastoid foramen.

INDICATIONS

This operation was first employed on patients who had failed to respond to treatment by other means and who had preexisting facial nerve paralysis. Patients with carcinoma of the parotid gland presenting with facial nerve paralysis have had an extremely poor prognosis. Eneroth[1] made this point in a review of 864 parotid tumor cases. He stated that persistent facial paralysis found preoperatively proved to be associated with a hopeless prognosis. None of their 12 patients with this finding survived longer than five years. The value of spontaneous facial nerve paralysis as a prognostic sign has been borne out by my personal experience, but further reference to this point has not been found in the literature. Spontaneous paralysis of the facial nerve is uncommon in the less highly malignant carcinomas of the parotid gland such as the acinic cell carcinomas and the adenoid cystic carcinomas. If only 12 such cases were found in the large series of Eneroth, then it might seem that the surgical problem which they present is a very rare one. Much of my work has been performed in an institution where cancer is often first encountered in far advanced stages, therefore a higher incidence is to be anticipated. Also, this operation is a valuable introduction to techniques of partial temporal bone resection. These techniques are extended somewhat beyond

classical radical mastoidectomy but are more limited in scope than the nearly total temporal bone resection described by Parsons and Lewis.[3]

At the present time the indications have been broadened to include not only those cases of carcinoma of the parotid gland with pre-existing facial nerve paralysis but also all highly malignant carcinomas of the parotid gland involving the posterosuperior portion of the gland and all malignancies of the pinna or external ear canal invading the parotid gland.

DIAGNOSIS

In most cases a clinical diagnosis of malignancy can be made with a high degree of accuracy before biopsy. This is based mainly on rate of growth and presence of facial nerve paralysis and fixation. Many of these patients have had recurrences following treatment by other means. In such cases we are often guided by the previous histological findings and the present clinical findings and may perform definitive surgery without biopsy. A superficially placed mobile nodule might first be treated by superficial parotid lobectomy. If on permanent sections the tumor on histological examination is found to be one of the more highly malignant forms, and it extends to the posterior capsule, then the more extensive operation is carried out as soon as possible. Deeply placed nodules are sometimes biopsied by open operation without superficial lobectomy. Some patients have had ulcerating lesions which were easily biopsied with forceps.

Sialography is sometimes helpful in establishing the nature of parotid masses but has not been of help in determining the posterosuperior extent of these lesions. Roentgenograms of the mastoids, temporomandibular joint and mandible are obtained on an individualized basis.

It is very important to examine the patient thoroughly to rule out a primary malignancy in other areas, such as the scalp or nasopharynx.

TECHNIQUE

General anesthesia with endotracheal intubation is used. The usual skin incision is indicated in Figure 2. This incision must often be modified. If skin is involved over the parotid gland, then the incision is carried in front of the pinna leaving it attached to the posterior flap in order to avoid compromising its blood supply when the involved area of skin is removed. If it is possible to save the skin of the ear canal and the eardrums, then the incision is most conveniently placed behind the pinna,

Figure 2. The usual skin incision.

since the skin of the external canal can be freed from behind much more easily. When parts of the pinna itself must be sacrificed, the flaps should be planned to minimize the residual deformity and to preserve a functional external auditory meatus. In this situation experience with the various types of mastoidectomy and tympanoplasty is invaluable.

A standard neck dissection is begun in the subclavian triangle and proceeds from the clavicle upward until the specimen is left attached only by the insertion of the sternocleidomastoid muscle to the mastoid bone, the digastric muscle, the internal jugular vein, and the fascial and areolar tissue attachments between this muscle, the parotid gland and the submandibular gland. If there are no lymph nodes that are grossly involved in the lower neck, a nerve graft is taken that includes the trunk of C-4 and three or four long supraclavicular branches; these branches are then split into six or eight bundles.

The sequence of illustrations shows how the pinna can be incorporated into the anterior skin flap with preservation of the skin of the ear canal skin and tympanic membrane (Figs. 3, 4 and 5). Above the insertion of the sternocleidomastoid muscle, the incision is made through the periosteum, and the periosteum is elevated anteriorly away from the bone. The removal of the ear canal skin and tympanic membrane is

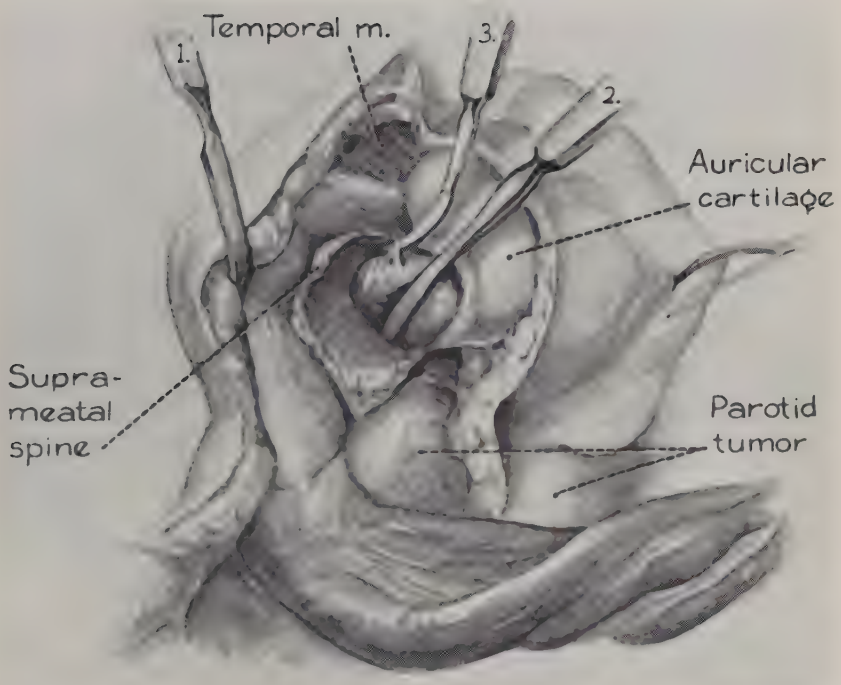

Figure 3. The first step in developing the skin flap of the cheek. *1*, Lempert elevator used to strip periosteum from outer cortex of mastoid. *2*, Canal skin elevator. *3*, Sickle knife used for sharp division of fibers extending into the tympanosquamous suture.

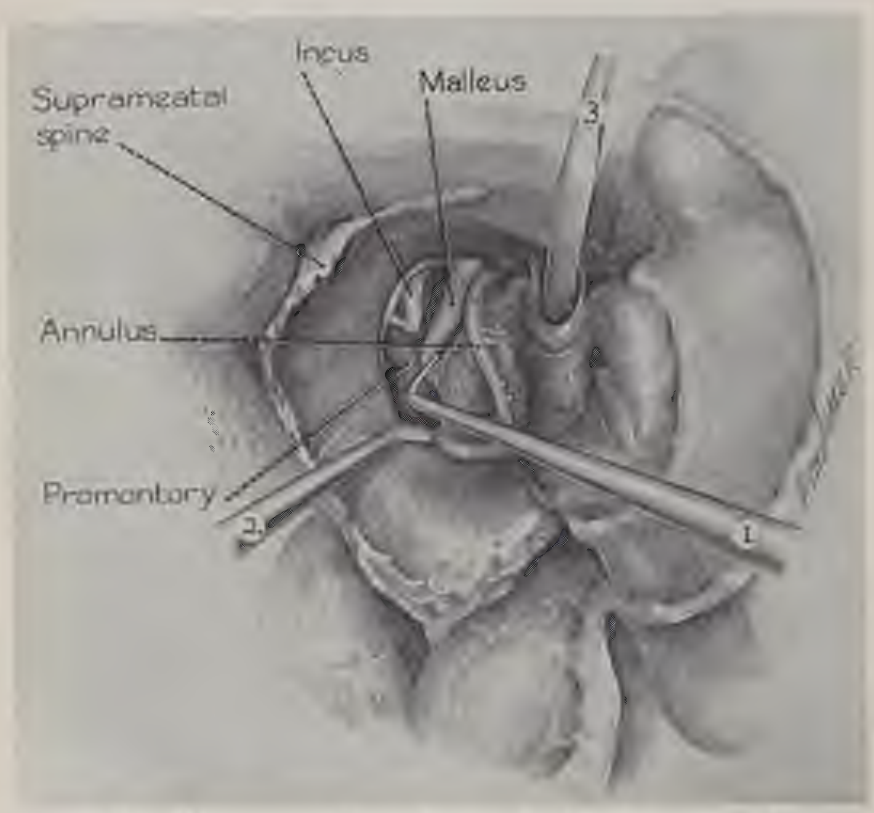

Figure 4. Freeing the tympanic membrane. *1*, Right angle knife used to divide mucous membrane along undersurface of malleus and remove tympanic membrane from it. *2*, Curved periosteal elevator used to free annulus. *3*, Canal skin elevator stripping skin from anterior bony external auditory meatus.

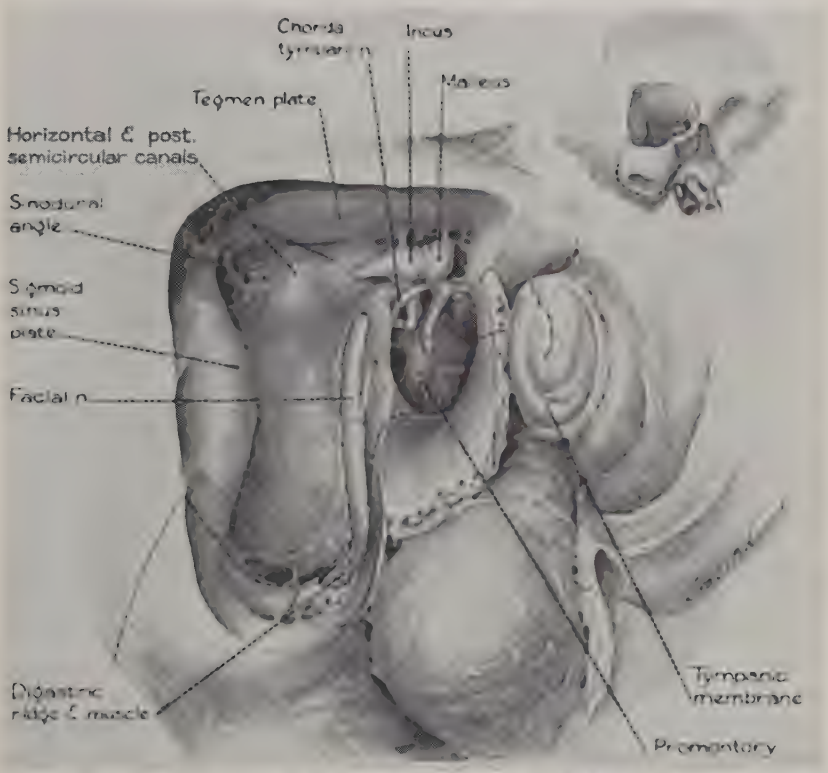

Figure 6. The mastoid cavity after exposure of the facial nerve. The dotted lines indicate sites of division of the facial nerve and bone of the mastoid and ear canal. The inset shows the area of bone removal viewed from its inferior surface.

aided by the use of an operating microscope. Incision through the mucosa on the undersurface of the malleus makes separation of the tympanic membrane from it relatively easy. Inadvertent perforations are most apt to occur near the anterior sulcus, and particular care should be exercised in the dissection of this area. After the skin of the ear canal has been completely freed, the skin of the cheek is elevated from the lateral surface of the parotid gland. The periosteum is then incised from the upper posterior border of the sternocleidomastoid upward to the temporal line. The periosteum is elevated away from the lateral surface of the mastoid except over the tip. An incision is made along the temporal line and continued along the upper edge of the zygomatic arch. Periosteum is also elevated along this incision for a distance of 2 to 3 mm.

Beginning at McEwen's triangle the mastoid cortex and air cells are removed until the sigmoid sinus plate, the digastric ridge, the tegmen tympani, the body of the incus and the head of the malleus are well defined (Fig. 5). The facial nerve is uncovered beginning at the digastric ridge and proceeding upward. After the descending portion has been completely exposed, the posterior bony meatal wall is removed with a rongeur, and the facial nerve is divided 3 to 4 mm. above its exit at the stylomastoid foramen. With straight Smith-Petersen osteotomes, the mastoid and external canal walls are divided as indicated in Figure 5. The bone is then fractured through the hypotympanum by gently prying with the osteotome. The parotid gland is then dissected away from the mandible and masseter muscle (Fig. 6). If necessary the masseter muscle and even portions of the mandible can be removed. Exposure of the deep lobe of the parotid gland and medial surface of the internal pterygoid muscle is excellent, however, without disturbing the mandible.

While the specimen remains attached by a sheet of fascia along its anterior and upper edges, peripheral branches of the facial nerve are dissected out and marked. The nerve graft is then sutured into position with 6-0 black silk (Fig. 7). A temporalis muscle flap is then turned down into the mastoid bowl to cover the nerve anastomosis. Two medium Penrose drains are inserted through stab wounds. I no longer perform suction drainage because of the frequent occurrence of fluid collections behind the clavicle with its use. The tympanic membrane is packed into position on the ossicles, and the skin flaps are closed. A layer of interrupted sutures is used in the platysma, and a continuous interlocking suture is used for closing the skin. A synthetic suture material (Tevdek) is employed using size 5-0 for the closure and for all ligatures except for those on major vessels, for which size 3-0 is used. A lateral tarsorrhaphy is often performed to protect the cornea pending return of facial nerve function. A pressure dressing is applied using knitted cotton gauze rolls to hold it in position.

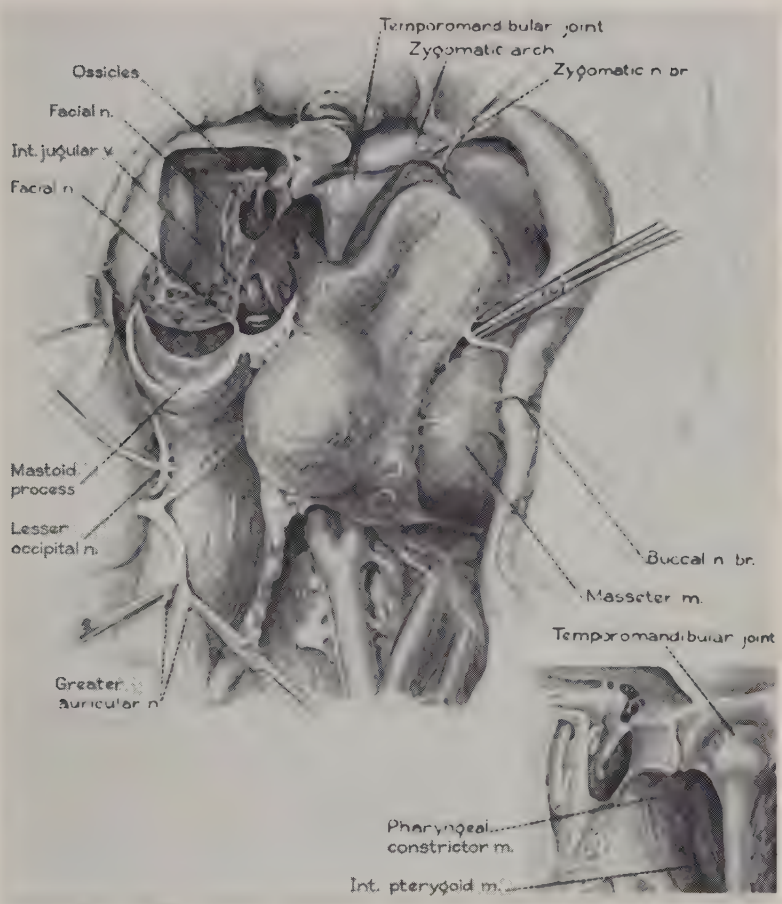

Figure 6. Removal of the parotid gland and isolation of peripheral branches of the facial nerve. For better orientation, portions of the neck dissection specimen are eliminated. The inset illustrates the improved access to the deep lobe of the parotid gland lying between the superior constrictor and internal pterygoid muscles.

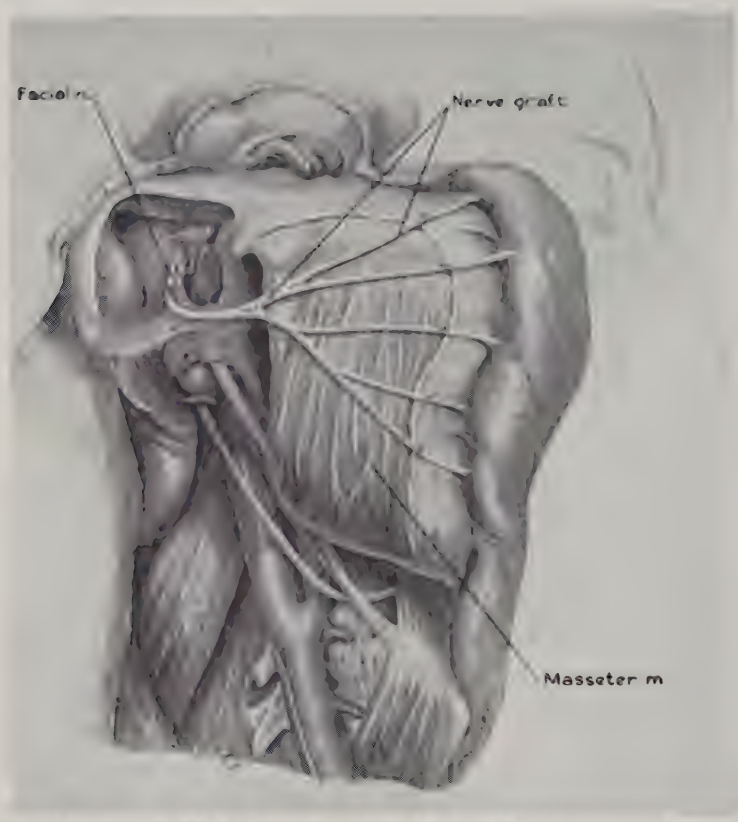

Figure 7. Removal of the specimen completed and facial nerve graft sutured into position.

CASE REPORTS

H.S., a 54 year old man, was admitted to Cook County Hospital April 16, 1962. He was semicomatose and therefore unable to relate his medical history. His brother stated that 3 years earlier a growth in the left cheek was biopsied and found to be malignant. The patient was treated by radiation therapy. The tumor did not respond to treatment, and he was admitted to a nursing home where he later developed a left facial nerve paralysis and slowly deteriorated. He spent all of the time lying in bed, lost weight and became incontinent. Review of his past history revealed that he was involved in an accident in 1919 and as a result required an operation for removal of his right kidney and another operation for fusion of his lower spine.

On examination the patient was found to be moribund, malnourished and incontinent. There was a small sacral decubitus ulcer. There was a 6-cm. hard mass in the left parotid region which was fixed to the underlying structures. A small scar beginning near the lobule of the ear was fixed to the outer surface of the mass. There was a complete left facial nerve paralysis. There were no palpable cervical lymph nodes. Blood urea nitrogen was 67 mg. per 100 ml. The electrocardiogram showed changes suggesting an old anteroseptal myocardial infarct. After blood transfusion and rehydration his blood urea nitrogen decreased and his state of consciousness improved.

On May 8, 1962, the parotid tumor was excised and left neck dissection performed using the technique described. The lobule of the ear with an adjacent ellipse of skin containing the biopsy scar and most of the masseter muscle were included with the specimen. In the early postoperative period there was a small dehiscence of the wound below the ear. This was repaired with split-thickness skin graft.

The microscopic sections were interpreted as showing a malignant mixed tumor, one of the more highly malignant varieties of parotid gland carcinoma. It has been over three years since surgery, and there is no evidence of recurrence. There has been no recovery of facial nerve function, but the patient is well adjusted to this handicap. He eats without difficulty, has no trouble with his eye, speaks with a normal voice and cares for himself.

T.B., a 75 year old man, was first seen at Northwestern University Medical School in the Otolaryngology Clinic on March 14, 1962. He developed "numbness" of the right side of the face and inability to close his right eye in January, 1962. On examination he was found to have weakness of the facial muscles on the right side, but there was at least some movement present in all muscle groups. Sensation on the face was normal. Taste was tested and was apparently not affected. A Schirmer test for lacrimation was normal. The audiogram showed a symmetrical high tone sensory-neural loss. A sialogram was technically unsatisfactory. No masses were felt in the parotid gland or neck; but, 3 weeks later, a firm but poorly defined nodule was felt deep between the tip of the mastoid and mandible. An open biopsy was performed, and a diagnosis of undifferentiated carcinoma was made.

After some delay the patient was persuaded to undergo operation. On June 29, 1962, he underwent the operation described. Healing was

uneventful. At follow-up on July 7, 1965, more than three years following operation, there is no evidence of recurrence. No facial nerve function has returned.

DISCUSSION

This operation has been performed for such a short period of time and has been applied to such a heterogeneous group of cases that meaningful statistics cannot be provided. It is believed nevertheless that good results in patients with highly malignant lesions, such as those cited, fully justify continuing our efforts. Several cases of carcinoma of the external ear have been treated with variations of this technique, and two cases of glomus jugulare tumor were treated by a variation in which the facial nerve was preserved but lifted out of its canal and no neck dissection was performed. All bone overlying the sigmoid sinus and jugular bulb was then removed in order that portions of these structures might be removed in the manner described by Hamberger and Gejrot.[2]

Although more time-consuming, this operation is not looked upon as being more radical than total parotidectomy with neck dissection, since the only disability that is likely to be added is a conduction type hearing loss, and the deformity is no greater. To date all nerve grafts in our experience have been unsuccessful in restoring facial movement. This is probably to be expected in most such cases where facial nerve paralysis has existed for long periods of time preoperatively, where the proximal anastomosis lies on avascular bone and the graft itself is long. There is no real disadvantage in attempting this reconstruction, however.

SUMMARY

A surgical procedure has been described which was designed to treat effectively those carcinomas of the parotid gland which extend into the regions near the ear canal and mastoid. In addition to providing for removal of a margin of normal bone around the posterosuperior capsule of the parotid gland, the operation gives excellent exposure of the deep lobe of the gland.

REFERENCES

1. Eneroth, C. M.: Histological and clinical aspects of parotid tumors. Acta otolaryng. (Stockholm) Supp. *191:* 1–99, 1964.

2. Hamberger, C. and Gejrot, T.: A contribution to the diagnosis of glomus jugulare tumors. Laryngoscope *74:* 1029–1036, 1964.
3. Parsons, H. and Lewis, J. S.: Subtotal resection of the temporal bone for cancer of the ear. Cancer *7:* 995–1001, 1954.

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Preoperative Irradiation and Bronchopulmonary Sleeve Resection for Lung Cancer

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In recent years there has been a rekindling of interest in preoperative irradiation in the surgical treatment of lung cancer. Bromley,[3] Bloedorn and Cowley,[2] and Paulson and Shaw[6] have probably reported the largest series of patients treated by the combined modalities. The latter two authors along with Johnston and Jones[4] have also introduced bronchoplastic procedures as a treatment of choice in certain instances of low grade malignant lesions without nodal involvement or in individuals in whom poor cardiac or pulmonary reserve demands a compromise procedure.

It is likely, however, that the pathologic situation permitting sleeve type or bronchoplastic procedures does not occur frequently (5 per cent of all resections in Paulson and Shaw's series[5]). Our group has been able to accomplish a sleeve type lobectomy in only seven patients having surgery alone in the past four years. In contrast, the use of preoperative supervoltage irradiation has permitted some type of sleeve type resection in 21 patients of the first 100 receiving both irradiation therapy and surgical resection.

This report consists of a series of 25 patients operated upon in a three-year period from August, 1962 to July, 1965. Admittedly, the follow-up of the recent patients is far too short, but it is hoped that tentative answers to the questions as to the feasibility of the lesser resection, the healing of anastomoses of irradiated bronchial tissues, and the survival of the earlier patients will be of value.

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PATIENT MATERIAL

Table 1 summarizes the age distribution as well as the sex incidence. The preponderance of male over female patients is readily noted. The occurrence in older age groups can be seen, and of the patients listed in the 70 to 79 decade, one was 77, one 74, and two 73 years of age.

In 24 of the 25 patients, positive diagnosis was obtained from sputum cytology, bronchoscopic biopsy material, or cell blocks of scrapings and secretions. The one remaining patient had several Class III Papanicolaou cytology reports and was subjected to preoperative therapy because of these reports plus the presence of a large mass seen on the chest roentgenogram. This lesion was eventually established as an adenocarcinoma and was one of two of this type in the series. There were two undifferentiated carcinomas and the remainder (21) were squamous cell types.

PREOPERATIVE IRRADIATION

In brief, the radiotherapy program has been carried out in two institutions where there is an interchange of personnel, but at the University of Illinois Hospitals, a betatron unit is used while Cobalt 60 is the method employed at Presbyterian-St. Luke's Hospital. The dosage and field of irradiation are similar, and in general this amounts to 4500 r given in approximately a four-week period. In the past two years, the two institutions have been participants in the National Institute of Health Preoperative Lung Cancer Study and have adhered to a somewhat standardized dose and field of irradiation.

Following completion of irradiation the period of delay before operation has been about four to six weeks, although an occasional patient has required a longer interval because of prolonged recovery from radiation sickness.

ANESTHESIA

An attempt has been made to simplify this aspect of the surgical procedure as much as possible in two respects. First, thiopental (Pentothal), hexafluorenium bromide (Mylaxen), succinylcholine and a mixture of nitrous oxide and halothane with oxygen are administered and varied as demanded by the patient's condition. Secondly, we have not used multilumen tubes for endobronchial anesthesia but have used a long Magill tube which is directed into the left main bronchus by the operator after the proximal bronchial division is accomplished on the right side.

Table 1. *Age and Sex Distribution of Patients*

	AGE			
	40–49	50–59	60–69	70–79
Male		8	11	4
Female	1	1		

The patient can then be maintained on left endobronchial anesthesia with inflated cuff and an essentially airtight system. This set-up is also utilized when right pneumonectomy with lower trachea and proximal left main bronchus resection is done. However, in two patients of this series the pulmonary artery and vein communications were maintained and a second tube and anesthesia unit were used with the tubing placed into the right lung through the thoracotomy wound.

Procedures on the left side require only a standard-length endotracheal tube; and, rather than cross-clamping the bronchus proximal to the anastomosis, we have utilized two or three Hibbs sponges sutured together and inserted tampon fashion into the main bronchus. Prior to ligation of the last several sutures these are removed and the knots tied, completing the anastomotic closure.

As an adjunct, one patient in this series required partial pulmonary bypass, utilizing a disposable plastic bag oxygenator. The details of this unique problem are the subject of a future case report but, suffice to say, the bypass was used when the remaining right lower lobe would not maintain the patient while left upper lobe–main bronchus anastomosis was in progress.

ANASTOMOTIC PROCEDURES

Figure 1 illustrates the four major types of procedures performed. One additional type of anastomotic resection was done in which the lower and middle lobes of the right lung were resected with high amputation of the right main bronchus. The amputated right upper lobe bronchus was then anastomosed to the stump of the right main bronchus.

The most frequent sleeve procedure employed in this series was the Type 1 illustrated in Figure 1. Thirteen patients received this type of resection modified in one of the group by the additional resections of the middle lobe and a sleeve of the right pulmonary artery. The Type 2 resection and anastomosis were accomplished in four patients and the

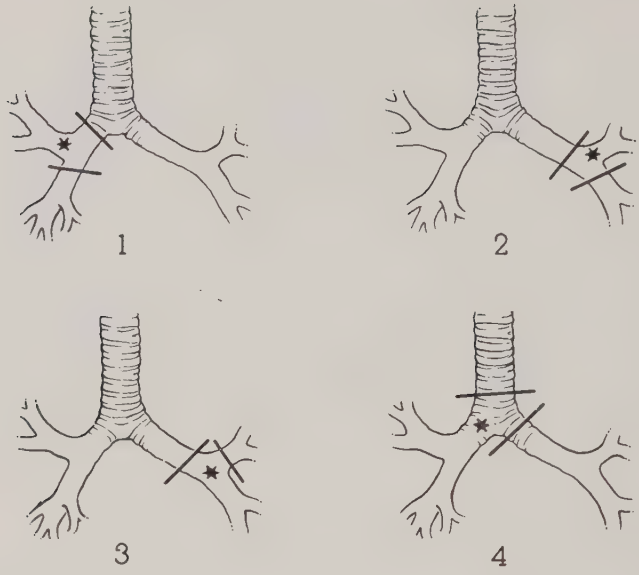

Figure 1. Major types of resection and anastomosis. Tumor site designated by asterisk.

Type 3 procedure in five. Only two patients required total high resection (Type 4) because of the location of the tumor. The lower end of the trachea, carina and proximal left main bronchus were excised with the right lung, with anastomosis accomplished between the end of the trachea and the left main bronchus.

SURGICAL PRINCIPLES

It is not within the scope of this report to give a detailed description of the operative technique, but it is important to emphasize the major principles of the procedures.

Of paramount importance is the initial endoscopic assay of the bronchial pathology. In almost all of the patients undergoing both radiotherapy and anastomotic resection, the repeat bronchoscopy procedure reveals a remarkably improved if not almost normal appearance. Accurate determination of the extent of the tumor initially will be a deciding factor in planning the extent of the sleeve resection. At the time of thoracotomy in the majority of patients, it is impossible to tell from frozen tissue studies of the margins of the sleeve resected whether tumor is present.

All of the resections were accomplished through a posterolateral

incision through the fifth interspace without resection of a rib. The patients were placed in a lateral position, turned approximately 30 degrees anteriorly with the upper arm extended in an exaggerated fashion to pull the scapula forward. An attempt to accomplish a left upper lobe sleeve resection through an anterolateral incision in one patient in the smaller, nonirradiated group convinced us of the superiority of the posterolateral approach. With the latter the anastomosis was angulated and stenotic, functioned poorly, and was corrected with a second procedure accomplished through a posterolateral approach.

As a general rule, the prior radiation has not appreciably added to the technical difficulty of the dissection. We have had an opportunity to do resective procedures in two patients after a longer period, 16 months and 18½ months following completion of therapy; and under these conditions, the tissue changes and technical difficulties are extreme.

The tissue changes encountered will be related to the degree of inflammation present before the therapy was given; and if extensive inflammatory reaction is observed in the initial x-ray films, the technical difficulties would be present if no radiation therapy were given.

The essence of these observations is the direct bearing on the feasibility of the sleeve type resection itself. The procedure cannot be accomplished unless the major bronchial and vascular components can be isolated and separated well above and below the proposed site of resection and anastomosis.

On the right side, it is necessary to separate completely the upper lobe from the lower and middle, isolate the artery and free it from the bronchus intermedius well below the upper lobe. The right main bronchus must be well separated from nodal and inflammatory tissues above and below the upper lobe take-off, identifying the proposed sites of division and anastomosis. Ordinarily, the venous channels are no problem if care is taken not to interrupt those branches on the superior surface of the middle lobe which actually are segmental veins in those situations of absent or poorly formed minor fissure. An example is seen in Figure 2.

On the left side, fissure completion is also necessary. One must be able to interrupt the arterial branches to the upper lobe without compromise of the main artery lumen and frequently coarctation clamps are required to occlude the branches flush with the wall of the artery continuing to the lower lobe. Division of the lower lobe bronchus or the upper lobe bronchus must be done with care because of the small margin of bronchus before the take-off of the superior or lingular segment respectively.

The anastomosis between the left lower lobe bronchus and the left main bronchus is quite similar to the procedure on the right after the

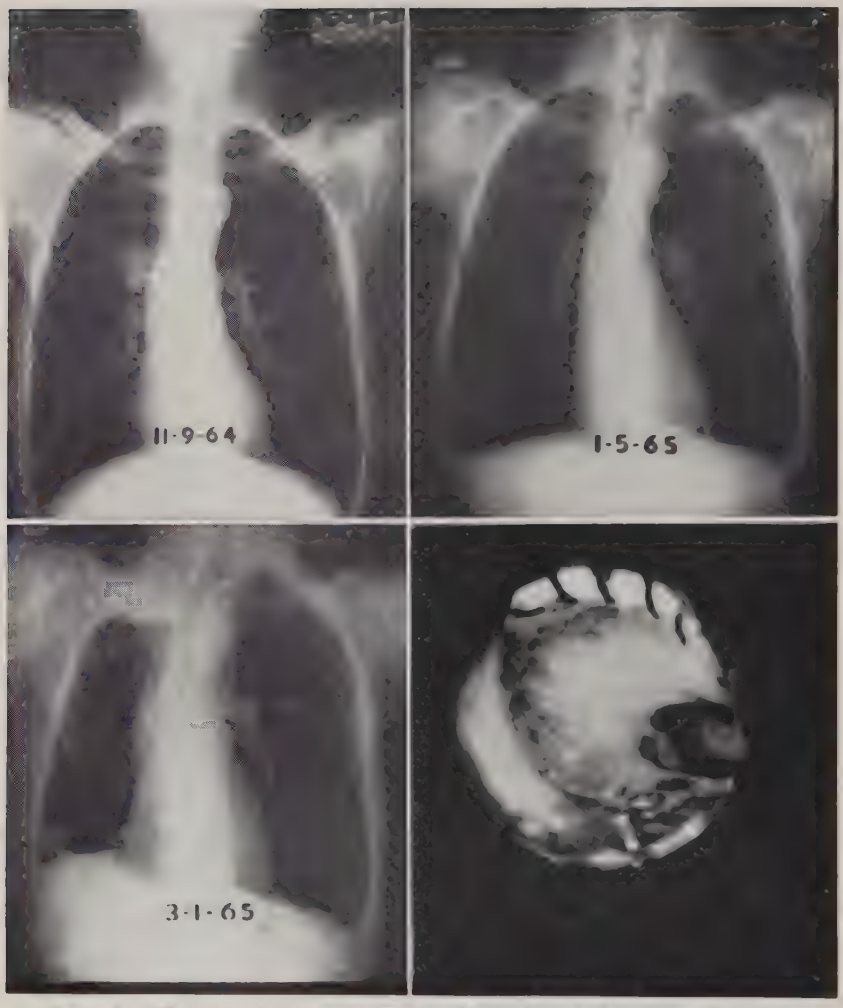

Figure 2. Chest roentgenograms: Preirradiation (11-9-64), postirradiation (1-5-65) and postoperative (3-1-65) of a 65 year old white male with epidermoid carcinoma, right upper lobe orifice. Endoscopic view of anastomosis (Type 1), demonstrating silk sutures.

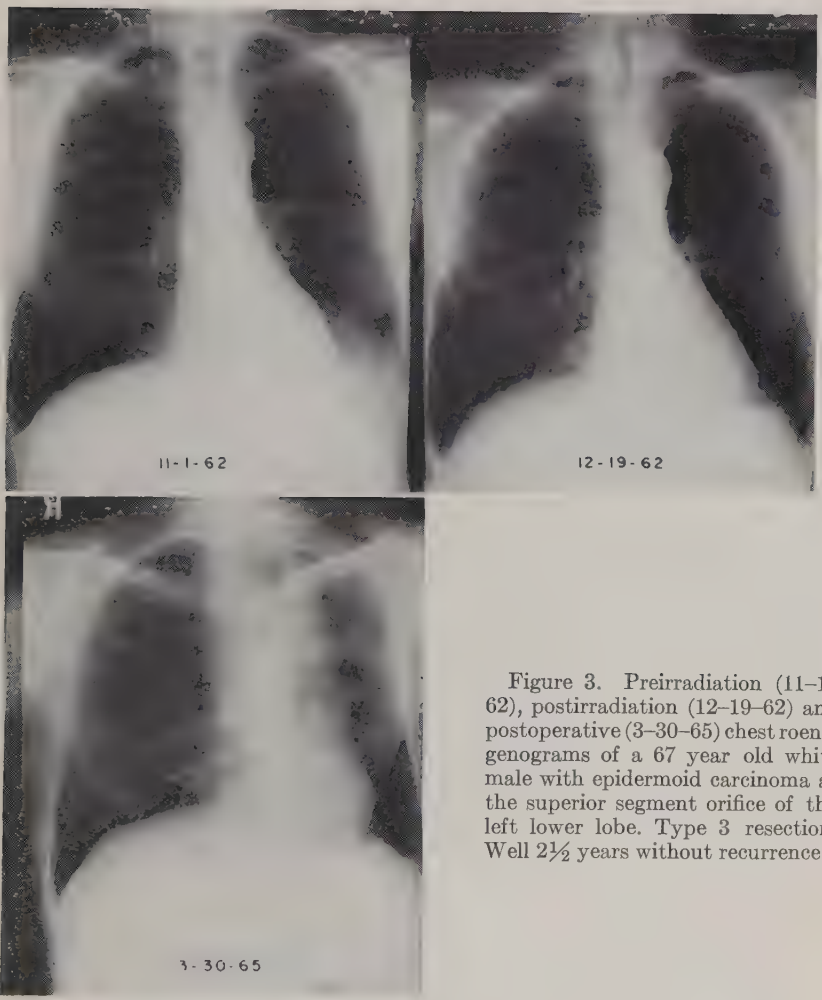

Figure 3. Preirradiation (11–1–62), postirradiation (12–19–62) and postoperative (3–30–65) chest roentgenograms of a 67 year old white male with epidermoid carcinoma at the superior segment orifice of the left lower lobe. Type 3 resection. Well 2½ years without recurrence.

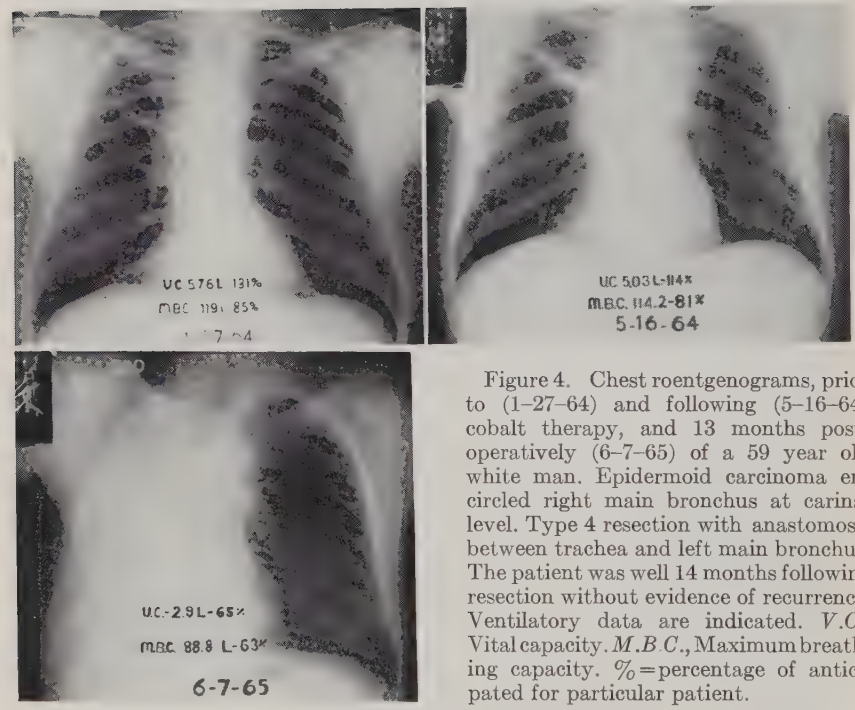

Figure 4. Chest roentgenograms, prior to (1–27–64) and following (5–16–64) cobalt therapy, and 13 months postoperatively (6–7–65) of a 59 year old white man. Epidermoid carcinoma encircled right main bronchus at carinal level. Type 4 resection with anastomosis between trachea and left main bronchus. The patient was well 14 months following resection without evidence of recurrence. Ventilatory data are indicated. *V.C.*, Vital capacity. *M.B.C.*, Maximum breathing capacity. % = percentage of anticipated for particular patient.

right upper lobe is removed. The anastomosis between the upper lobe and the main bronchus on the left poses a more difficult technical problem because of the position of the artery (Fig. 3). It is advisable to place at least six sutures anteriorly before they are tied to allow for more accurate approximation of the bronchial lumen. It is quite difficult to retract the artery to expose the bronchial ends if the first suture placed is ligated.

In tracheobronchial anastomoses (Fig. 4), it is necessary to isolate and free up the lower trachea, left main and right main bronchi. Because of the reaction involving the azygos vein, it is usually interrupted posteriorly and flush with the superior vena cava.

In all of the anastomotic procedures, there is a disparity in the lumina of the two bronchi. In general, cartilage-to-cartilage approximation is carried out, stretching the membranous portion of the smaller lumen and allowing for folding of the larger lumen so that the difference is equalized.

Until recently, interrupted through-and-through 3-0 silk sutures have been used, placing the knots on the outside of the lumen of the bronchus. Granulation tissue reaction to varying degree, in some instances requiring bronchoscopic removal, has developed. This disadvantage has been almost completely eliminated by the use of plastic suture material in the last three patients undergoing sleeve resections and anastomosis.

The importance of bronchial aspiration must be emphasized. The operator should thoroughly aspirate the bronchi of the salvaged lobe or lobes before the anastomosis is started and again before the final sutures are tied. It is advisable to aspirate the main bronchus of the dependent lung through the proximal main bronchial opening. This is easy on the right but somewhat more difficult when left-sided procedures are done unless the point of division is fairly high on the left main bronchus.

Protective cover of the anastomotic suture line is probably more necessary in patients having had prior irradiation than those without. It is our practice to rotate a large flap of parietal pleura and suture it to the bronchus after the flap is placed to envelop the suture line.

We believe that the anastomotic line endobronchially behaves as a barrier until mucosal regeneration is completed, and routinely subject the patient to bronchoscopy on the first or second postoperative day. This is done with extreme care, usually passing the instrument to a point above the anastomosis and inserting a soft catheter beyond to remove secretions. This may be repeated, but if the problem cannot be handled usually by two aspirations a tracheostomy is done.

The patients with tracheobronchial anastomoses had tracheostomies performed immediately at the completion of the procedure.

RESULTS

Analysis of our efforts combining irradiation and sleeve resection are directed toward complications (Table 2) and survival data (Table 3).

Initially, a problem of major concern was the question of healing of the irradiated, anastomosed bronchi. Actual separation occurred in only two patients, the first of whom had a right upper lobe sleeve resection. This patient had been discharged two weeks after her procedure and readmitted two weeks later because of episodes of hemoptysis. Failure to demonstrate any bronchial separation or fistula on repeated bronchoscopy led us to delay intervention and the following day the patient had an exsanguinating hemorrhage. Approximately one-half of the suture line had separated when the tissues were examined at autopsy. The other partial separation occurred in the one patient who had the right upper lobe anastomosed to the right main bronchus. When this patient developed episodes of hemoptysis and a collapse of the right upper lobe, she was re-explored promptly and the pneumonectomy completed.

One patient had a space infection. This did not communicate with the anastomotic site, but rather developed at the apex of the right pleural cavity. It was treated subsequently by open thoracotomy drainage.

Narrowing of the anastomotic site due to excessive granulation tissue was a problem in four patients. Two of these had undergone right upper lobe sleeve resections, one being the first patient in the study. This patient required repeated dilatation procedures over the course of the following year. The last dilatation was done over two years ago, and the patient is well without recurrence of his cancer just short of three years since his sleeve resection lobectomy.

The second patient had similar stenosis following an identical procedure. In this individual, a contributing factor was thought to be the tension on the suture line where the anastomosis was accomplished. This patient was one of the few in whom a positive diagnosis of tumor was obtained on frozen section at the distal lumen. It was necessary to remove approximately 3 cm. of bronchus, dividing the distal bronchus immediately above the right middle lobe. The stenosis and granulation tissue give no evidence of recurrence one year after the surgical procedure.

Excessive granulation tissue formed in the second of the two tracheo-

Table 2. *Complications*

Operative mortality	1
Anastomotic dehiscence	2
Empyema	1
Suture granulation and stenosis	4

bronchial anastomoses. This patient has had several endoscopic procedures to remove these formations in the six months that have elapsed since his operation.

The first of the two tracheal sleeve resections resulted in very little granulation tissue reaction, however, and the endoscopic views show a widely patent tracheobronchial lumen (Figs. 4 and 5). Similarly, the endoscopic view in Figure 2 reveals minimal granulation tissue reaction although this photograph was taken approximately two weeks following surgery.

The last patient with stenosis had firm scar tissue narrowing at the anastomotic site between the left lower and left main bronchus. Tumor recurrence was a factor in this problem and survival was only eight months following the procedure.

It is hoped that, with the excellent results noted in three recent cases in which plastic suture material was used, the granulation tissue problem can be eliminated or at least minimized.

The one operative death occurred, unfortunately, in the operating room. This patient developed cardiac arrest just prior to the beginning of the anastomotic procedure. Both the anesthesiologist and the surgical team failed to realize the nature of the anesthesia difficulties and slowly increasing anoxia. The dissection was particularly difficult with pronounced bleeding which demanded prolonged, complete attention. It was later shown that the bevel of the anesthesia tube had impinged on the lateral wall of, first, the trachea and, later, the medial wall of the left main bronchus.

Two other deaths occurred within a month following the surgical

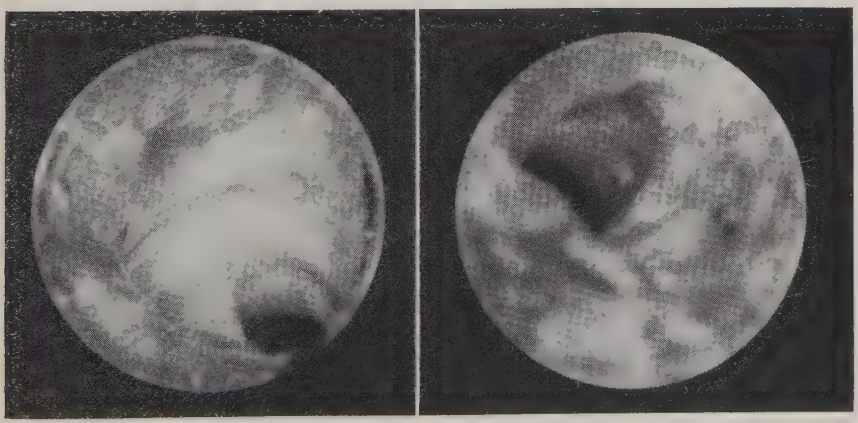

Figure 5. Tracheobronchial anastomosis following right pneumonectomy and carinal resection. *Left:* Bronchoscope at suture line. *Right:* Bronchoscope at midtracheal position.

Table 3. *Survival Data*

	OPERATIVE PROCEDURE			
	Rt. U.L. Sl.	Lt. U.L. Sl.	Lt. L.L. Sl.	Trach. Sl.
Patients	13	4	5	2
Early deaths	3	0	1	0
Late deaths	1	3	0	0
Alive with tumor	2	0	1	0
Alive without tumor	7	1	3	2

procedure. One patient had been discharged for over a week and had been seen at a clinic visit at which chest film studies were made the day before his sudden death. The other patient succumbed to bronchopneumonia in the anastomosed lobe approximately one month postoperatively. This patient had left the hospital against medical advice, refusing further bronchoscopic procedures postoperatively.

These four patients thus represent the early deaths indicated in Table 3, which summarizes the survival data of the total series. One dehiscence failure is not listed, as the patient was eliminated by virtue of the pneumonectomy completion. She lived only six months, however, succumbing to a widely disseminated carcinomatosis.

The survival times of the four patients representing "late deaths" were 3 months (right upper sleeve), 5½ months, 8 months and 16 months (left upper sleeves).

Three patients alive but with recurrent tumor had procedures done 15 months and 5 months (right upper sleeves) and 18 months (left lower sleeve) earlier.

In addition to these three, there are 13 additional survivors without evidence of recurrence of cancer ranging from several months to three years. Seven patients of this group have survived more than two years. (Seven of nine patients had their procedures more than two years ago.)

Patients who have completed preoperative irradiation demonstrate a different picture at the time of thoracotomy. Tissue changes in the area of tumor as well as the lymph nodes make it difficult for any analysis of the procedure as to what degree of compromise occurs by reason of its lesser magnitude. The residual nodal tissues infrequently reveal tumor tissues and not uncommonly the lung tissue itself may be "tumor sterilized." The Maryland investigators[1] pointed out that this latter effect was by no means a criterion of cure. Our cases have been analyzed as to the presence of residual tumor. In the seven patients alive without tumor recurrence after more than two years, only three were "tumor sterilized,"

the remaining four demonstrating residual tumor in the pulmonary tissue, with only one of these having regional node involvement as well. In contrast, of the seven patients representing late deaths or tumor recurrence, all had residual tumor in the lung tissue and one of these had involved regional lymph nodes as well.

DISCUSSION

Both the reported series of sleeve type procedures of Johnston and Jones[4] and Paulson and Shaw[5] lend concrete support to the validity of such localized resections in the treatment of lung cancer. The former authors had 7 of 15 patients alive for more than two years while the latter investigators reported 6 of 14 patients alive more than four years. Indeed, in our small group of nonirradiated patients, three of four patients are alive over two years.

Although a factor accounting for the small number of nonirradiated patients treated by sleeve resection may have been a reluctance to be conservative in the face of a malignant problem, the major reason was probably the unsuitability of the patient material for this type of resection.

The fact that utilizing supervoltage therapy preoperatively allowed a sleeve type procedure in over 20 per cent of the first hundred patients treated would suggest the importance of the irradiation in converting patients to sleeve resection candidates.

At the present time, the final conclusions of the combined preoperative lung cancer study have not been reached although preliminary figures suggest there is no added value provided by the irradiation therapy beforehand. Very likely the value of the modality resides in its selective use, its application in hilar lesions in an attempt to convert an individual to a sleeve candidate being one of a few valid reasons for it.

The surgical complications experienced in our group of patients compared favorably with the reported series. The problems of dehiscence and suture granulation stenosis should be minimized if not eliminated by the pleural envelope over the anastomosis and the use of plastic suture material.

There is a tendency, however, for the development of a slowly increasing fibrosis in this group of patients, and this becomes more apparent with longer survival (see Fig. 3). The chest x-ray in the patients surviving two to three years show this gradual loss of lung volume in the tissues salvaged by the anastomosis. In comparison, the few patients having this procedure without irradiation still demonstrate a well-expanded lobe or lobes after two or three years.

This then may be an undesirable side-effect which will have to be

weighed against the advantages. Function studies reveal a slight diminution in capacity with longer survival time and this is not seen in the patients without irradiation. However, the capacity still seems to exceed that of patients undergoing total resection.

Although comfort and capacity cannot be minimized, longer survival will still be the sought-after criterion of value. Some investigators are coming to the conclusion that no increased survival will obtain using irradiation preoperatively, and this may eventually be shown by comparing irradiated and nonirradiated series as in the controlled, double-blind, randomized study now in progress.

With regard to our own small group, the survival of 13 of 25 patients would seem to be a very acceptable figure, especially when seven of the first nine patients are alive two or more years and free of recurrent carcinoma.

SUMMARY

Twenty-five patients with lung cancer who had completed a course of supervoltage preoperative irradiation were treated by pulmonary resection combined with sleeves of the bronchial tree. In two of these pneumonectomy was done along with resection of the lower trachea, carina and a portion of the left main bronchus, accomplishing anastomosis between the left main bronchus and trachea.

There was one operative death, two instances of anastomosis dehiscence, one of empyema, and four of excessive granulation tissue reaction to sutures requiring repeated bronchoscopic dilatation and suture removal.

Three additional early deaths after discharge from the hospital were attributed to suture line separation, coronary heart disease and bronchopneumonia.

Late deaths occurred in four additional patients from 3 to 16 months postoperatively and were attributed to disseminated malignancy.

Three patients alive from five months to 18 months have recurrent metastatic carcinoma but 13 patients of the 25 are living without recurrent cancer for periods from several months to three years. Seven of this survival group represent the salvage among nine patients operated upon more than two years ago.

The combination of irradiation and pulmonary sleeve resection is a justifiable therapeutic program, creating no increase in surgical complications or mortality and providing very acceptable survival data.

The major advantage of preoperative radiation may well be the conversion of patients into candidates for sleeve resection.

REFERENCES

1. Baker, N. H., Cowley, R. A. and Linberg, E.: A follow-up in patients with bronchogenic carcinoma "locally cured" by preoperative irradiation. J. Thoracic & Cardiovasc. Surg. *46:* 298 (Sept.) 1963.
2. Bloedorn, F. G. and Cowley, R. A.: Irradiation and surgery in the treatment of bronchogenic carcinoma, Surg. Gynec. & Obst. *111:* 141, 1960.
3. Bromley, L. I. and Szur, L.: Combined radiotherapy and resection for carcinoma of the bronchus: Experience with sixty-six patients. Lancet *2:* 937, 1955.
4. Johnston, J. B. and Jones, P. H.: Treatment of bronchial carcinoma by lobectomy and sleeve resection of the main bronchus. Thorax *14:* 48 (March) 1959.
5. Paulson, D. L., and Shaw, R. R.: Results of bronchoplastic procedures for bronchogenic carcinoma. Ann. Surg. *151:* 729 (May) 1960.
6. Paulson, D. L., Shaw, R. R., Kee, J. L., Mallams, J. T. and Collier, R. E.: Combined preoperative irradiation and resection for bronchogenic carcinoma. J. Thoracic & Cardiovasc. Surg. *44:* 281 (Sept.) 1962.

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Surgery of Acquired Disease of the Small Arteries

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The operative techniques that have been developed for the treatment of acquired small vessel arterial disease are all sufficiently recent to be considered new. Such surgical procedures, performed principally to reconstruct the local vascular lesion of arteriosclerosis, have undergone refinement and maturation over the past decade. Not only has there been continuing reappraisal of the technical aspects of such surgery, but also an evolution as regards surgical diagnosis, investigation and indications.

In this paper we will offer our current judgment on the surgical management of patients with small vessel disease, discussing the problems of cerebrovascular, coronary, abdominal aortic branch and lower extremity occlusive disease. Preoperative as well as operative considerations are given, as in some instances the most important recent changes have been in the application or indication for operation rather than technical revision of the procedure itself. The matters of practical importance are recorded as prompted by review of day-to-day practice on a busy cardiovascular service. Emphasis is thus dictated little by theoretic considerations but rather by what the newer thinking is in the areas where it is most often applied. Thus, cerebrovascular disease is given much greater emphasis than coronary atherosclerosis. Space does not permit completeness and surgical technique is discussed only when a particular point is to be made.

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CEREBROVASCULAR DISEASE

Among the newer procedures performed by the cardiovascular surgeon are those designed to increase the cerebral blood flow. The realization that a major percentage of strokes have their basis in obstruction of the extracranial portion of the four arteries serving the brain serves as a basis for these procedures. The four cardinal symptoms encountered in patients with extracranial stenotic or occlusive disease are: (1) recurrent dizziness or instability; (2) numbness, awkwardness, weakness or paralysis in an extremity; (3) transient visual blurring or loss, especially if unilateral, and (4) deterioration of ability to think, remember and do mental work. Other complaints, less frequently encountered, include transient aphasia, a mass in the neck, or even the declaration by a patient that he can hear an intermittent sound which we recognize as his arterial bruit. The importance of these symptoms in serving as a warning of the disaster that may follow the inevitable deterioration of arterial blood flow is well recognized by the vascular surgeon, but probably is yet to be fully appreciated by the profession as a whole. Symptoms of basilar artery insufficiency produced by vertebral obstruction are considerably less common than the cardinal complaints listed above, which infer the presence of the most usual brachiocephalic lesion; namely, carotid bifurcation stenosis. Indeed, we find that the majority of patients with symptoms suggestive of vertebral basilar insufficiency have multiple lesions affecting the carotid as well as vertebral systems.

Clinical Examination

As with most lesions involving the cardiovascular system, physical examination in patients with brachiocephalic disease has proved most illuminating and diagnostically rewarding. Two-thirds or more of the patients with such lesions will have abnormalities revealed to the careful examiner. Obstruction of the major arteries at their origins from the aortic arch, or lesions of the innominate bifurcation are always detectable using the palpating fingers and stethoscope. Diminished, delayed or absent peripheral pulses betray the presence of common carotid, subclavian and innominate obstruction. Significant stenosis of these vessel origins, such as to compromise blood flow, invariably produces a systolic bruit and often a thrill. Palpation of the carotid bifurcation often permits definition of the arterial division as a hard, irregular, noncompressible atheroma-containing structure. A thrill over the bifurcation is not often present, but in the majority of patients with local atheromatous lesions, a bruit will be heard. Whereas the loudness of the bruit has been observed to bear no relation to the degree of arterial narrowing, the duration of the bruit is of interest. If the bruit is both systolic and diastolic, often mim-

icking the sound of an arteriovenous fistula, then one can predict extreme stenosis of one or both of the bifurcation branches. Unlike the patients with a proximal stenotic lesion at the level of the arch which invariably is associated with a bruit, a sizeable number of patients with bifurcation stenosis have no bruit to be heard. Therefore, such patients with suggestive symptoms must be studied with arteriograms, whether or not physical findings are present.

Of particular interest to us on review of our experience with physical examination in patients with brachiocephalic disease are those patients shown to have the so-called "steal syndrome." Such patients have obstruction of one of the major branches of the aortic arch, usually the left subclavian artery, and much less often the innominate or right subclavian channel. Collateral blood flow in such a situation may be derived in part through reversed flow in the vertebral artery or, on infrequent occasion (when the innominate is occluded), even the common carotid itself. In such a situation a collateral wrist pulse is usually palpable, but it is small, delayed and plateau in character as compared to the opposite normal radial pulse. If high grade stenosis rather than occlusion exists at the innominate or subclavian origin, then a bruit will betray this upon examination of the affected channel distal to its origin.

If a "steal syndrome" exists, and in the usual case the vertebral artery is furnishing collateral flow to the upper extremity vascular bed distal to the obstructive subclavian origin, this fact can be shown in over 90 per cent of such patients by the application of a simple test. One examiner monitors the collateral wrist pulse and a second occludes transiently one of the common carotid arteries. If as a result of this abrupt diminution in total cerebral flow the reversed flow in the vertebral artery sharply decreases, the wrist pulse will be abolished. A positive test on one side and a negative one on the other infers that on the side of no effect the internal carotid may be obstructed or narrowed, thus contributing little to cerebral blood flow. The fact that the test is so often positive, and the truth that collateral upper extremity blood flow is so readily affected by changes in but one of the collateral channels—the vertebral, attests to the important deficit in total cerebral blood flow that can result from proximal subclavian artery occlusion.

Arteriography

The advisability of arteriography and choice of its type are dependent on the history and physical findings. In general, patients with the above-mentioned cardinal symptoms suggestive of cerebral ischemia should be studied by percutaneous carotid arteriography if no other explanation can be found for their complaint. Patients with symptoms suggestive of basilar artery insufficiency should have both vertebral and

carotid bifurcation visualization. The latter is important because in our experience many apparently symptomatic vertebral lesions will have an associated carotid bifurcation stenosis and, indeed, relief of symptoms may well occur following eventual repair of the carotid lesion. Isolated vertebral artery obstruction is quite uncommon in our series of patients, and we feel that isolated vertebral disease is an unusual cause of cerebrovascular insufficiency.

In patients with symptoms of cerebral ischemia and positive physical findings, the angiographic studies are directed to the type by these findings. The majority of patients will have evidence of carotid bifurcation lesions and percutaneous carotid injections under local anesthesia are done. The smaller group of patients with stenosis of the origin of the aortic arch branches are usually subjected to arch angiography, although our experience indicates that physical examination is about as reliable as arteriography in determining the locale and degree of proximal arterial narrowing. The physical findings in vertebral origin stenosis are unreliable, and visualization of these vessels is indicated principally by suggestive symptoms. We have found that the practical approach to subclavian origin occlusion with the "vertebral subclavian steal" syndrome is through well programmed carotid angiography. The reason is that no x-ray study is required to disclose the obvious arterial occlusion of the aortic arch branch; carotid bifurcation lesions are commonly associated and require definition, and last, that carotid injection of dye with proper follow-through on serial films may demonstrate the "steal." Total obstruction of the common carotid never requires arteriography for definition, for the presence of such a lesion is conspicuous on physical examination.

An evolution has slowly occurred in our judgment about patients with positive physical findings, usually a carotid bifurcation bruit, and yet no ischemic symptoms. Initially, such patients were not subjected to study, but with increasing experience with angiography and the surgical treatment of the lesions demonstrated, our thinking has changed. At present we consider that a carotid bifurcation bruit discovered in an asymptomatic patient who is not over age 70 and who generally has a good prognosis for longevity should be studied. Although carotid arteriography has been shown in our center to have a very low incidence of morbidity and mortality, we have the impression that the risks may be greater in the very aged.

Carotid arteriography should be done in such a way as to permit minimal morbidity and mortality and to visualize as well the intracranial vascular tree. The latter is important to permit discovery of the patient with intracranial occlusive disease and also to reveal the occasional non-arteriosclerotic intracranial lesion such as berry aneurysm, neoplasm or vascular malformation. Biplane technique obtaining serially programmed

films will permit total posteroanterior and lateral visualization with a single small injection of contrast media. It is important that the injection be made low in the common carotid to avoid needle disruption of the atheroma.

Indications for Operation

Selection of patients for elective surgery is done according to the following principles. Patients having carotid bifurcation lesions narrowing the internal carotid lumen by 50 per cent or more are subjected to endarterectomy. An occasional patient will be seen who presents with clinical symptoms suggestive of multiple small cerebral emboli and who should be operated upon if the x-rays show a ragged, possibly ulcerated carotid bifurcation. Completely occluded internal carotid arteries are not operated upon, as early experience showed that the chronic obstruction could not be surgically relieved. Patients with tortuous and possibly kinked internal carotid arteries are operated upon only when clinical symptoms reinforce the significance of the radiologic finding. A select group of patients with chronic complete occlusion of the internal carotid and stenosis of the external carotid have been operated upon. These patients have usually had bilateral internal carotid obstruction and the angiogram has demonstrated the narrowed external carotid to be contributing through collaterals to intracranial blood flow. Generally, such individuals are quite symptomatic and the surgical effort is directed toward compensating for their not directly remedial cerebral ischemia.

The smaller group of patients having vascular obstruction other than at the carotid bifurcation include those with common carotid origin stenosis, and subclavian or innominate stenosis or occlusion. Repair is undertaken in patients with significant common carotid stenosis, but those with complete occlusion of the common carotid are usually inoperable, since the internal carotid in its extracranial portion will be thrombosed. An occasional patient will maintain internal-external carotid flow and thus bifurcation patency. This unpredictable state can be revealed only by exploration, which is occasionally indicated in the symptomatic patient when there is no other available approach to augmenting cerebral arterial flow. Patients with chronic occlusive lesions of the subclavian origin seldom have significant arm ischemia and operation is usually done for associated cerebral ischemia. Curiously, the less common right subclavian occlusion is more apt to produce hand ischemia than left-sided lesions. On occasion upper extremity ischemia, with of without central nervous system symptoms, will serve as an indication for arterial reconstruction. We have not recommended operation to patients with vertebral subclavian steal who are asymptomatic.

Individuals with isolated symptomatic vertebral artery stenosis, as previously mentioned, were seldom seen in our series of patients. Most often the symptomatic vertebral lesion was associated with carotid disease. In such an event the carotid lesion should be repaired first and a later judgment made about vertebral surgery. Often following the carotid reconstruction the patient will become symptom-free. In the event that the vertebral artery narrowing is associated with inoperable carotid disease (as complete internal carotid occlusion) or if the surgeon can satisfy himself that the isolated vertebral narrowing is genuinely producing symptoms, then a direct approach to vertebral repair is in order.

Surgery of Acute Strokes

In the above paragraphs we have considered the recent thinking about elective management of patients with cerebrovascular disease. Of particular interest to us has been a problem of a somewhat different sort—the management of the patient who is seen during an acute stroke. Our approach to such patients has clearly undergone change, as we have reviewed our surgical results. All patients with extracranial disease whom we have seen and operated upon, or considered for operation during their acute stroke, have had carotid artery occlusion. We have not seen a patient with an acute stroke due to isolated vertebral or subclavian obstruction. At present we divide patients suspected of having acute strokes due to extracranial vascular lesions into three groups based on their neurologic state.

Group I includes those who are having repetitive small strokes with recovery or near recovery between the rapidly occurring attacks. These patients have varying degrees of hemiparesis, but rarely have hemiplegia. When first seen, such a patient is observed for recovery from his transient neurologic deficit, but when the progressive and repetitive character of the course is apparent, then arteriography and surgery are indicated. Results of carotid endarterectomy have been excellent in this group, even though operations in many instances were performed on confused, aphasic and partially paralyzed patients.

Group II includes those patients with fixed neurologic deficit who are often aphasic and usually are hemiplegic. Hemiplegia and an unchanging neurologic residual are, in our experience, indicative of cerebral infarction. These patients, if the cause of the stroke is an extracranial arterial lesion, usually have either carotid bifurcation thrombosis or, less often, cerebral emboli from an ulcerated carotid atheroma. Emergency surgery in this group can often restore blood flow through the diseased carotid bifurcation but should usually not be undertaken. Results of surgery reveal on review that most patients are not benefited by the

procedure and the remainder fall into two small groups of about equal size. These latter patients with about equal chance will have either a dramatic improvement or will suffer cerebral hemorrhage into the now pressure-perfused cerebral infarct and may die. To summarize surgical results in this group with cerebral infarct, one can say that most are not benefited, a few are rendered worse, and an exceptional patient is clearly improved. When considering such a patient during his acute stroke, we generally defer arteriography a matter of days until neurologic recovery has reached a plateau. Arteriography will then most often show complete occlusion of the internal carotid artery, but occasionally the irregular ulcerated atheroma already referred to. As significant bilateral carotid artery disease exists in at least 20 per cent of patients, the artery opposite the side of greatest suspicion is also carefully studied. Although the only inflexibly applicable rule is to "individualize" judgments about these patients, in general we attempt to wait four to six weeks for the recent cerebral infarct to stabilize and heal before an elective operation is performed to augment cerebral blood flow.

Finally, Group III includes a small number of patients in whom there is reason to believe a stroke has occurred on the basis of extracranial disease and in whom the magnitude of the event and its effect on the patient are such as to make useful survival unlikely. Isolated instances of dramatic salvage may be obtained through carotid endarterectomy even though in the given case the odds are much against success. We have had, for example, an experience in which emergency carotid endarterectomy was done on a totally unresponsive patient who was supported in his collapse with vasopressors and a respirator, and who following operation recovered to return to full-time employment.

The Technique of Surgery

The technical aspects of brachiocephalic arterial repair have reached their present state of satisfactory development through a process of revision and improvement. Initially, many reports appeared describing techniques, especially ones to protect cerebral integrity during arterial reconstruction. Many of the latter were complicated and time-consuming, such as those embodying local or systemic hypothermia for protection of the brain against transient ischemia. Our current methods are characterized by simplicity, rapidity of execution and very satisfactory final results.

Over three-fourths of the operations performed are for arteriosclerotic narrowing of the internal carotid artery origin. Carotid endarterectomy may be done under general or local anesthesia, and the choice is based on analysis of the individual patient. Most are performed under

general anesthesia, but the cooperative elderly patient with acutely symptomatic, high grade internal carotid stenosis and associated serious systemic disease may be operated upon most safely while awake and then be permitted to resume activity soon afterward.

During exposure of the common carotid and its two branches, particular care is taken to avoid trauma to the diseased bifurcation, as undue manipulation and squeezing may cause cerebral emboli and subsequent postoperative neurologic deficit. Fifty milligrams of heparin are given intravenously to the patient, clamps applied to the three carotid channels, and a longitudinal arteriotomy performed beginning in the common carotid and extending through the atheroma into the internal branch. A tapered, flange-tipped, especially constructed polyvinyl shunt is then placed in the internal carotid above, and the other larger end in the common carotid below. The shunt is held in place by ring forceps of appropriate design which correspond in size to the smaller and larger ends of the shunt. The shunt is made sufficiently long to permit it to loop gracefully between its proximal and distal insertions, thus allowing it to be laid conveniently out of the way on either side of the arteriotomy. The presence of the shunt does not interfere with or render difficult the endarterectomy. Since this technique is simple and maintains normal cerebral perfusion, we routinely use it.

After removal of the atheroma, the residual arterial wall is carefully inspected for minute, loosely adherent tissue fragments and the upper extreme of the dissection is examined to be certain the intima is secure and safe from the potential of dissection. If the atheroma is completely removed in its internal carotid extension, the upper intima may require tacking and this must be done if it is thickened or loosely adherent. The arteriotomy is then approximated from both ends with a continuous fine suture until the presence of the shunt prevents further closing. The shunt is then withdrawn, the vessels occluded with clamps and the final few stitches applied to close the arterial opening. Immediately prior to the last stitch the external carotid is opened and the flow of blood retrograde displaces all air from the repaired segment. Release of the common carotid clamp causes the initial flow to pass into the external branch. Finally, the internal carotid is released. Direct closure of the artery has proved very satisfactory and patch graft angioplasty is rarely used. Absolute attention to meticulous detail, unhurried because of the security provided by the shunt, produces consistently good results making surgically induced neurologic complications infrequent. Postoperative anticoagulation is not used. It is our practice to repair the artery with the most disease first and then, in the instance of bilateral carotid artery stenosis, to perform opposite endarterectomy in five to ten days' time.

Vertebral artery origin stenosis is treated by autogenous saphenous

vein patch angioplasty which guarantees a generous orifice. The operation is done through a cervical incision above and parallel to the clavicle.

Arterial narrowing at the common carotid origins and subclavian or innominate stenosis or occlusion are customarily treated by the bypass graft technique. Most often the prosthesis is proximally anastomosed to the thoracic aorta and the limb or limbs appropriately sutured end-to-side to the channel to be revascularized beyond its diseased origin. Alternative techniques include bypass grafts between the carotid and subclavian arteries in the neck when there is impaired flow in one and normal flow in the other on the same side of the neck. A crimped dacron tube 10 mm. in diameter has been used for bypass procedures, with the use of a bifurcation graft when more than one major brachiocephalic artery is to be repaired. Endarterectomy with few exceptions has proved to be less reliable and unnecessarily difficult for the management of these proximal lesions. When repairing an obstructed subclavian artery origin it is appropriate to inspect the nearby vertebral origin and correct any stenosis that may be present.

Symptomatic patients with the "vertebral subclavian steal syndrome" are treated by revascularization of the subclavian artery, usually by a prosthetic graft from the adjacent descending thoracic aorta to the subclavian beyond its atheromatous origin. We have considered vertebral ligation as a means of increasing available cerebral blood flow in a few instances of symptomatic "steal" in elderly poor-risk patients. The latter idea seemed attractive since it could be done quickly through the neck under local anesthesia and would be certain to improve cerebral if not upper extremity circulation. All such patients to date have had conventional repair of their "steal" without mortality or regret, but the procedure remains one available for rare indication.

CORONARY ATHEROSCLEROSIS

It would be impossible to consider the surgery of small artery acquired disease without commenting about coronary atherosclerosis. Certainly, in terms of incidence and clinical importance, coronary artery disease ranks high on the list and deserves equal discussion with extracranial arterial disease affecting blood flow to the brain. In practice, though, the surgical approach to these lesions of the small arteries serving the myocardium is not anywhere nearly so effective and operations on them are uncommonly done. This remains true despite very important recent advances in coronary angiography, which of necessity had to precede any widespread and practical surgical approach to coronary insufficiency. The problem and obstacle to surgical relief of coronary disease remains

the fact that the disease involves vessels of very small caliber, often in a diffuse manner, in a place that is surgically not easily accessible and where mortality from attempted direct repair remains high.

At present three types of surgical approaches are employed at the University of Illinois and Presbyterian-St. Luke's Medical Center: (1) coronary endarterectomy; (2) indirect myocardial revascularization, principally the method of Vineberg using the internal mammary transplantation, and (3) left dorsal sympathectomy for symptomatic relief of anginal pain. We have accepted for study only individuals affected at an unusual early age, or those with disabling symptoms which were intolerable despite medical management. Although all three types of procedures have been undertaken, the total experience has been small.

Coronary endarterectomy is recommended only under the circumstances of a highly favorable local lesion of a main coronary artery in a patient with severe disability and who is in a relatively younger age group. The procedure is done with total cardiopulmonary bypass and a fibrillating heart. A recent successful endarterectomy was facilitated by the use of an autogenous venous patch to enlarge and insure patency of the relatively small endarterectomized coronary. The Vineberg procedure has been described by him and we have performed it according to his technique. Left dorsal sympathectomy is not a new procedure for totally disabling anginal pain, but continues to give reliable relief when applied in highly selected cases.

INTRAPERITONEAL SMALL VESSEL SURGERY

During the last few years an increasing number of surgical procedures have been performed on the branches of the abdominal aorta. Two types of procedures have been done most frequently: operations on the obstructed renal arteries for relief of the Goldblatt phenomenon, and procedures designed to relieve intestinal ischemia. These will be discussed together.

Although Goldblatt produced hypertension experimentally in 1934 by renal arterial constriction, the medical profession only recently has interested itself in the spontaneous occurrence of such a cause of hypertension. Even today, the true incidence of renovascular hypertension is not known and probably is greater than realized. Any lesion which affects either total renal arterial blood flow or pulse pressure may cause the release of renin by the affected kidney and thus hypertension. Four types of lesions are encountered: (1) renal artery stenosis, (2) renal arterial thrombosis or embolism occurring without total renal infarction, (3) renal artery aneurysm, and (4) renal arteriovenous fistula. By far the most

common of these is renal artery stenosis, for which arteriosclerosis and fibromuscular hyperplasia are the two most usual among the dozen or so reported causes. Renovascular disease is to be particularly suspected in certain clinical situations, such as hypertension of recent onset, but as a practical matter it can be said that it should be considered in any hypertensive patient whose blood pressure elevation is such as to be productive of sequelae and is unfavorably influencing prognosis for life and well-being. In general, renovascular hypertension tends to be systolic and diastolic and is refractory to the usual medical management.

Patients with diminished arterial supply to the intestinal tract may have "intestinal angina" or "intestinal claudication." Typically, the arteriosclerotic narrowing of the celiac or superior mesenteric arterial origin results in postprandial abdominal distress, diarrhea, malabsorption and weight loss. The abdominal pain following meals may be so severe as to cause the patient to avoid eating, with accelerated weight loss. Owing to failure of fat absorption by the ischemic intestine, the patient may observe that his stools float. The syndrome may be easily confused with other varieties of gastrointestinal or biliary disease.

Physical examination in patients with disease of the abdominal aortic branches is seldom diagnostic. Abdominal bruits are listened for, but if found their significance must be shown by subsequent study. Nonsignificant bruits created by atheromatous change in the wall of the aorta or due to the often present tortuosity of the hepatic artery may be heard in clinically well patients.

Indirect evidence for renovascular disease may be gained through the timed nephrogram, intravenous pyelogram, radioisotope renogram, and differential renal excretory studies. Screening studies for intestinal ischemia include negative cholecystographic and gastrointestinal barium studies, plus absorption studies performed with fecal analysis. Ultimately, though, the precise definition of aortic branch vascular disease is obtained by angiography. In our experience, the best technique of angiography employs retrograde femoral catheter passage and then injection of the contrast media during multiple programmed film exposures. It is essential that studies designed to evaluate the celiac and superior mesenteric vessels include lateral views of the aorta.

Reconstruction of the renal vessels is undertaken to relieve hypertension and preserve or restore kidney function. Revascularization of the intestine is done to relieve the described symptoms and prevent bowel infarction from occurring as the diminished arterial flow further deteriorates. Surgical procedures designed to restore normal arterial perfusion may take varied technical forms such as endarterectomy, division and aortic reimplantation, patch graft angioplasty, resection and anastomosis, or bypass grafting. Numerically, both for renal and intestinal arterial

reconstruction, the most common procedure has employed the bypass graft technique using synthetic tubes. In the instances of celiac reconstruction it may be effective and most convenient to attach the distal end of the graft to the splenic artery, which in turn retrogradely supplies the other branches. These grafts from the aorta to the recipient vessel are short, subject to no motion, and are possessed of high flow rates—all factors which have contributed to the very satisfactory technical results.

Relief of intestinal ischemia can be regularly anticipated following a well conceived and executed reconstructive procedure, but recent review of our patients with surgically treated renovascular disease shows that only 52 per cent of hypertensive patients are actually cured at the end of one year or more of follow-up. An additional number of hypertensive patients are improved.

ARTERIAL DISEASE OF THE LOWER EXTREMITIES

Arterial insufficiency affecting the lower extremities may follow one of several anatomic patterns of vascular involvement: (1) aortoiliac, (2) superficial femoral, (3) distal small vessel disease below the knees, and (4) combinations of these. Isolated, principal involvement of the femoral or popliteal artery proper is unusual. Two questions are to be answered during the evaluation of a patient with ischemic legs: (1) which arteries are obstructed or narrowed, and (2) what is the resulting degree of ischemia. In particular, it is determined whether the problem is one of functional impairment only or there is actual or threatened tissue loss.

The history of intermittent claudication allows dependable diagnosis of arterial insufficiency and its degree and distribution give a clue as to its anatomic locale and severity. Acute or chronic progression to ischemic symptoms at rest is an ominous sign and demands of the vascular surgeon a maximal effort for limb salvage.

Physical examination permits definition of the anatomy of the obstructive arterial disease and allows objective analysis of the severity of the distal ischemia. Critical evaluation of the pulses, auscultation for bruits, and direct palpation of the arterial walls where accessible enables the examiner to predict the place or places of arteriosclerotic change. Upon examining the feet, trophic changes are looked for as well as cutaneous moisture, indicative of an active sympathetic nervous system.

Despite the precision that can be developed in physical examination, arteriography is nevertheless important. Aortic or femoral arteriographic studies are done depending upon the defined area of occlusive disease. Such x-rays confirm clinical judgment and by virtue of delayed exposures give detail of the arterial blocks, show their length, and, of great impor-

tance, outline the distal bed beyond the occlusion. Angiography must not replace intelligent bedside examination, but the information gained from it should be used to supplement inspection, palpation and auscultation. For example, an aortogram provides a view in only one plane of the aortoiliac system, and palpation of the femoral pulses often permits more precise judgment about the degree of functional stenosis and impairment of flow.

Decisions as to the proper mode of therapy are made on the basis of (1) a total assessment of the patient, (2) the anatomy of the arterial disease, and (3) the patient's degree of disability and ischemia. Each patient is to be considered separately and the tendency to treat the arteriogram is avoided.

In general, the more optimal the lesion in terms of certainty of a favorable immediate and late result from arterial reconstruction, the less severe can be the patient's degree of disability and objective ischemia to properly indicate the need for surgery. The less favorable the character of the arterial block for confident repair, the greater the need for patient disability and objective ischemia before deciding upon operation. In situations in which the problem amounts to limb salvage from amputation, very unfavorable anatomic lesions are properly pursued surgically. Sympathectomy remains a simple, safe and effective operation for ischemic disease, either as an alternative to reconstruction or an addition to it.

The treatment of isolated aortoiliac arteriosclerotic occlusive disease is well established and such patients undergo reconstructive surgery with a high probability of immediate and long-term success. Prosthetic grafts function very satisfactorily and there has been greater tendency in recent years to use them instead of endarterectomy. Endarterectomy is now used only in ideal candidates in whom the disease is highly localized to the aortic bifurcation and probably gives no better long-term results than bypass grafting. An effort is made to keep the reconstruction within the pelvis, but the original concern over passing the grafts under the ligaments to the femoral artery has not been justified.

Patients with superficial femoral artery disease are treated when reasonably short segments are involved and there is good run-off patency by saphenous autogenous vein bypass or, less often, by endarterectomy. If the distal bed is in any way compromised by disease, sympathectomy is also done, as it augments a successful grafting procedure and protects against the consequences of early or late graft occlusion. Prosthetic grafts are used only for long segments of occlusion, generally from groin to below knee, and then only in the instance of limb salvage operations. In our experience with prostheses in this region, their long-term function has been uncertain. Because prostheses are used in the most advanced cases, often with distal tissue breakdown, sympathectomy is usually

done also. Patients unsuited for autogenous vein technique, but whose nutrition is well preserved, are most often treated by sympathectomy because of its ability to protect the distal skin against ischemic change.

We have had little success in reconstructing small vessels distal to the popliteal bifurcation, and we extend bypasses to them or attempt an endarterectomy only in limb salvage situations. Sympathectomy is useful except in certain diabetics who have, by virtue of their neuropathy, autosympathectomized themselves.

Patients with multiple areas of occlusive disease are treated in varied ways, depending upon the disease pattern and their degree of ischemia. Over the last few years, we have come to appreciate how very effective the so-called "profunda revascularization" procedure is, and now in instances of combined aortoiliac and superficial femoral occlusion we usually obtain good results with it. The proximal aortoiliac element is treated with a bypass graft, restoring normal profunda perfusion, and with added sympathectomy we do not repair the usually extensive superficial femoral occlusion. The once often performed aortopopliteal grafts are now rarely done, as the profunda revascularization is adequate to maintain distal nutrition and has a much more favorable result over the long term. An occasional patient with well localized superficial femoral disease may be properly treated with aortic bifurcation prosthetic grafting, and an autogenous saphenous graft in the thigh. Patients with widespread disease who either lack a major element of aortoiliac disease or who have unsuitable profunda "run-off" at the groin, and yet who have well preserved skin on their feet, are treated by sympathectomy. Each patient is different and must be so assessed.

The technical performance of these procedures will not be described as the methods are reported elsewhere. The aspect of recent interest and the matter of current importance is the evolution of their application.

SUMMARY

Current surgical diagnosis, clinical investigation, operative indications, and in some instances surgical technique have been discussed in relation to problems of acquired occlusive disease of the smaller arteries. Our present thinking and practice in relation to such disease of the brachiocephalic, coronary, abdominal aortic branch and arterial supply to the lower extremities have been described.

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New Operations in Children's Cardiac Disease

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Congenital heart disease encompasses a wide spectrum, from the neonate who succumbs in the early stages of life to the rare elderly individual who has difficulties with the deterioration of age. In between lie the patients with patent ductus arteriosus, coarctation of the aorta, or intracardiac defects of the atrial and ventricular septums and pulmonic stenosis which have proved to be correctable with the perfection of the heart-lung bypass. The elderly patient with a congenital heart defect does not need help from the cardiovascular surgeon. It is the early or difficult lesion that benefits most from the new methods of correction or palliation.

Each decade brings a new phase in cardiac surgery; in the 40's the extrapericardial lesions were stressed; in the 50's with extracorporeal circulation great advances were made in the correction of septal

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defects; in the last several years, numerous innovations and many modifications of older operations have been devised in the treatment of the more complex lesions. The newest tool to support standard surgery in infants with cyanosis or low systemic flow is hyperbaric oxygenation.[3] Major activities in infant intracardiac surgery and in the development of exquisite cardiorespirator technique were reported at a recent symposium in Chicago.[6] Here we will present new operations from our clinic and other institutions as they aid the pediatric patients with difficult cardiologic problems.

SEPTAL DEFECTS ASSOCIATED WITH PULMONARY HYPERTENSION

Septal defects, whether atrial, ventricular or aortopulmonary, can be classified according to their effect on the pulmonary circulation. The more benign forms of these lesions do not produce significant pulmonary vascular changes and present little problem in correction. Those defects which produce the most severe pulmonary hypertensive changes and are generally considered inoperable will be discussed in a later section. The vast area in between includes defects which produce mild to severe changes in the pulmonary arterial vasculature, yet are clearly associated with a significant left-to-right shunt. Direct correction of these septal defects with severe pulmonary hypertension has led to an increased mortality; however, gradual closure of the defect appears to be better tolerated, and the use of a perforated prosthesis has been helpful in the repair of septal defects in such circumstances.[2, 5, 7] Eventually complete closure occurs. Recently, this method has been applied to the large aortopulmonary defect by Reis[17] in a patient with equal systemic and pulmonary arterial pressures and balanced right-to-left and left-to-right shunts. This patient's defect was closed with a perforated Teflon patch, the ideal size of the multiple perforations appearing to be 1.5 to 2.0 mm. in diameter. With a normal coagulation time one would expect closure to occur over a several-month period.

Infants with ventricular septal defects often develop a kinetic pulmonary hypertension with recurrent heart failure due to excess of flow and a small volume pulmonary vascular bed. Based on the work of Muller and Dammann[15] in 1953, main pulmonary artery constriction has been generally used and produces excellent palliation in many of these patients with complete correction of their defect performed at a late date. Some surgical groups are performing complete open heart correction as the initial procedure.[11, 12, 19]

Right Pulmonary Artery Banding

There is a group of patients who have severe pulmonary hyper-

tension with approximately equal pulmonary and systemic flows with little or no shunting. Banding the main pulmonary artery has not proved feasible in this type of patient, the heart deteriorating either during the operation or in the immediate postoperative period. Studies in our laboratory have indicated a progressive decrease in pulmonary vascular resistance associated with an increase in pulmonary vascular volume following depulsation of the pulmonary artery flow. Studies on unilateral pulmonary artery coarctation suggest that these changes may be beneficial in the patient with a limited pulmonary vascular flow-volume status. These and other findings[14] have led us to banding the right main pulmonary artery in patients with the extreme form of pulmonary hypertension. This technique has been used in nine patients. One early and one late postoperative death have occurred.

The operative procedure is fairly simple, with all bandings being done on the right main pulmonary artery just behind the superior vena cava. The degree of constriction must be determined by trial and error. A dacron tape 1 cm. wide is passed around the right main pulmonary artery to produce the occlusion. Pressure measurements are then taken proximal and distal to the band and are recorded as the band is tightened. Absence or marked depression of the pulse wave and a 10-mm. to 15-mm. decrease in the mean pulmonary artery pressure across the band is generally all that can be tolerated by the heart. These criteria of constriction usually occur with a 70 to 80 per cent lumen narrowing.

The postoperative period can range from benign to severe. Generally, those under ten years of age and with only one major intracardiac defect do well, but older patients and those with complex lesions have a violent postoperative course. Arrhythmias constitute the prime myocardial hazard, requiring judicious medical therapy. Table 1 indicates the patient's age, preoperative and postoperative measurements and diagnosis. The two deaths involve patients with a functional single ventricle and at present we are not performing the operation in this group. The classical cardiac silhouette and the type of pulmonary vascular disease found in this type of patient are demonstrated in Figures 1 and 2.

This patient (M.L.P.) was 10 months old when first seen. A clinical diagnosis of congenital heart disease with ventricular septal defect was made. She was then lost to follow-up for 16 years. Upon her return, the diagnosis was changed to Eisenmenger's disease. Minimal cyanosis was evident. Five years later the concept of unilateral pulmonary artery banding developed, with the intent to protect one lung damaged by hypertension from further insult. This concept was applied to this patient and the right pulmonary artery was constricted to 70 per cent of the original diameter, resulting in a decrease in pressure distal to the band. The pulse wave pattern was flattened. The postoperative course was stormy, marked by arrhythmias and cyanosis. Three years later her follow-up studies showed 66 per cent of total function in the "protected" lung by bronchospirometry, but catheterization and

Table 1. *Hemodynamic Data— Right Pulmonary Artery Banding*

PATIENT	AGE (yrs.)	RIGHT VENTRICULAR PRESSURES	MAIN PULMONARY ARTERY PRESSURES		RIGHT PULMONARY ARTERY PRESSURES (POST BANDING)		DIAGNOSIS	STATUS
		Range	Range	Mean	Range	Mean		
M.L.P.	21	94/2	94/49	64	78/46	58	Eisenmenger's complex	Alive
K.B.	10	75/10	75/45	59	68/48	54	"	Alive
R.W.	18	120/12	120/60	70		50	Corrected transposition (Functional single ventricle)	Deceased
D.G.	7	96/6	98/62	82			Eisenmenger's complex	Alive
A.J.	8			100		70	"	Alive
C.V.	12	110/0	105/65	80	70/65	60	"	Alive
S.W.	3 mo.		70/20	40		16	Complete transposition (Functional single ventricle)	Deceased
E.J.	8	110/0	Prior 110/60		RUL 73/63	66	Eisenmenger's complex	Alive
			Post 105/65	83	RLL 73/63	66		
S.P.	6	100/0	100/60	75	70/50	55	"	Alive

lung biopsy (tolerated without difficulty) do not show any significant changes. Pulmonary angiography demonstrates marked right pulmonary artery stenosis (Fig. 3).

The activity change with these patients is extensive. They are almost able to keep up with their contemporaries in the usual everyday activ-

Figure 1. (Patient K.B.). Typical postero-anterior and lateral chest x-rays of a patient with pulmonary hypertension. The prominent main pulmonary artery and decreased peripheral vascular markings are evident.

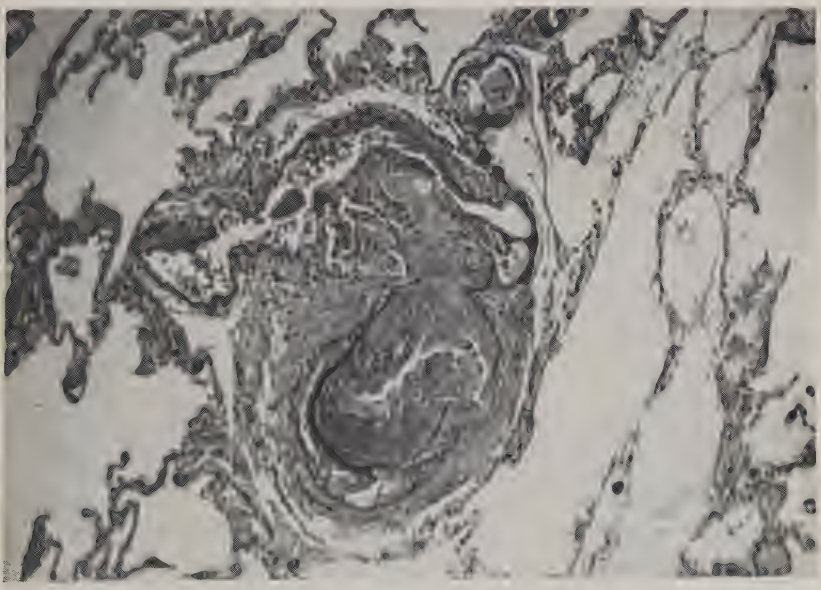

Figure 2. (Patient K.B.). Microscopic appearance of a biopsy of the lingula. Severe intimal proliferation and minimal medial hypertrophy are present in the arteriole.

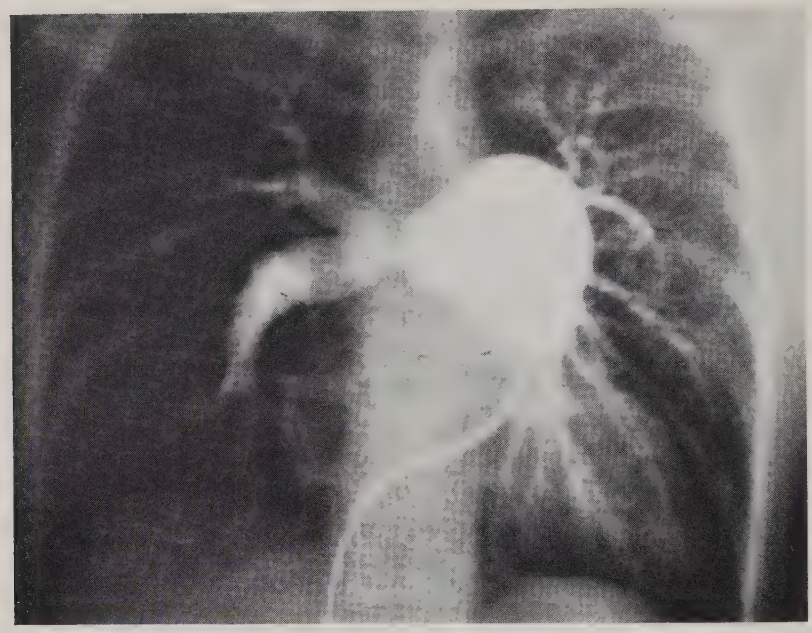

Figure 3. (Patient M.L.P.). Pulmonary arteriogram demonstrates marked constriction of the right main pulmonary artery.

ities. All patients have had a marked decrease in the frequency and severity of respiratory tract infections. Despite clinical improvement, further documented study will be needed over longer intervals to show a definite measurable improvement allowing the intracardiac defect to be corrected.

COMPLETE TRANSPOSITION OF THE GREAT VESSELS

This anomaly still vexes the physician. These small, underdeveloped, cyanotic youngsters do not tolerate the usual shunting procedures as pulmonary hypertension is often an inherent problem in the disease. The first successful total correction of this anomaly was accomplished by Senning.[18] His method of transposing the atrial septum and using the external anterior atrial wall as a flap is a difficult surgical procedure, but when successful produces complete correction. Recently Mustard[16] developed an atrial baffle using pericardium for complete correction. This operation presupposes the presence of a large atrial septal defect or artificially created defect produced in the Blalock-Hanlon procedure.[4] Autogenous pericardium is used to form the baffle. After cardiopulmonary

bypass is initiated an anterior atriotomy is made. The pericardium is then sutured around the pulmonary vein orifices and closed to exclude the orifices of the cavae and coronary sinus, diverting them into the posterior atrium and ventricle. Using this operation, five of seven complete corrections have been accomplished by Mustard.

PULMONARY ATRESIA WITH NORMAL AORTIC ROOT

This rare anomaly, if left untreated, has an 80 per cent mortality by by the age of six months.[10] A patent ductus arteriosus is necessary for life and its closure is probably the cause of the early mortality. Numerous shunting procedures such as the Potts or Blalock or synthetic grafts between the aorta and pulmonary artery have been tried, but have not proved successful over long periods of observation.

Combined Use of Caval and Systemic Shunts

In one recent case in which a combination of caval and systemic shunts was used, a remarkable increase in oxygenation was produced. Although unsuccessful because of an acute arrhythmia, it should be attempted again.

Patient M.F. a 24 months old girl, was admitted to the hospital dyspneic, cyanotic, underdeveloped and never having walked. Cardiac catheterization and

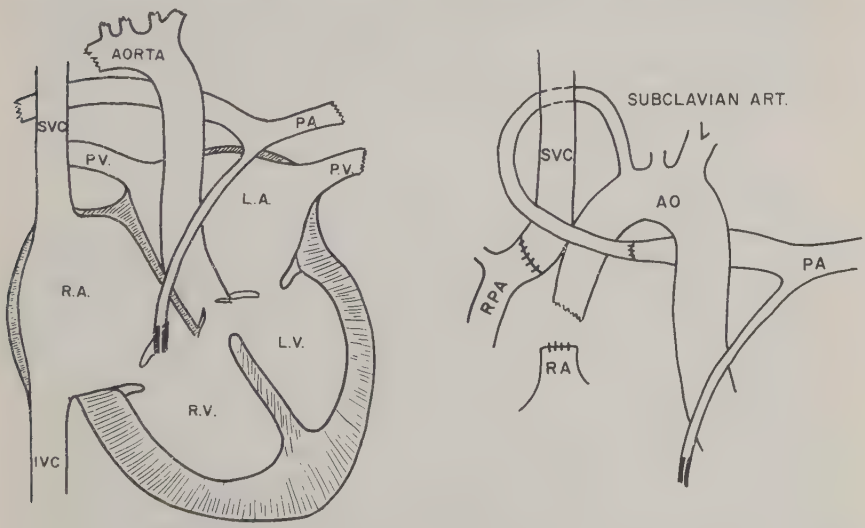

Figure 4. (Patient M.F.). *Left,* Outline of main defects. Pulmonary artery is minute. *Right,* Operative correction: Superior vena cava to right pulmonary (Glenn procedure) and right subclavian artery to proximal right main pulmonary artery anastomoses.

cinefluoroscopy led to the diagnosis of pulmonary atresia or hypoplasia with right aortic arch (Fig. 4, *left*). Via a right thoracotomy a Glenn procedure was done, joining the superior vena cava with the distal right main pulmonary artery. The right subclavian artery was then anastomosed to the proximal divided end of the right main pulmonary artery (Fig. 4, *right*). There was an obvious increase in systemic oxygen saturation at the completion of the anastomosis. The first 24 hours postoperation were unremarkable. However, early on the first postoperative day the patient developed a sudden arrhythmia and could not be resuscitated. Autopsy showed a patency of the anastomoses, minute main pulmonary artery, a normal right ventricle, ventricular septal defect, and enlarged bronchial arteries.

The use of the Blalock type anastomosis to the entire left lung may not produce an irreversible pulmonary hypertension and, combined with the Glenn procedure, could allow for long-term survival. This patient demonstrates the danger of arrhythmias to patients with cyanotic congenital heart disease.

LESIONS OF THE TRICUSPID VALVE

The tricuspid valve is only infrequently involved by acquired heart disease, but it is often the site of anatomic derangement in congenital heart disease. Congenital tricuspid valve lesions are of two types: (1) stenosis or atresia, and (2) displacement with or without deformity.

Total Correction of Tricuspid Atresia

Narrowing or absence of the tricuspid orifice impedes the flow of blood into the lungs. An intracardiac septal defect, generally atrial, must be present for the blood to return to the systemic circulation. These children are pathetic; they are cyanotic and they fail to thrive. The usual palliative procedure is a superior vena cava right pulmonary shunt. This allows for correction of the upper trunk venous return but does not aid the great inferior vena caval return flow and does not cure the child. Persistent cyanosis indicates a large right-to-left shunt which exposes the patient to all the dangers of polycythemia, cerebral abscess and systemic embolism. The following case history presents a method for total correction of tricuspid atresia.

Patient S.B. was a cyanotic 18 months old female patient when first seen at the University of Chicago clinics. Work-up studies established a diagnosis of tricuspid atresia, atrial and ventricular septal defects and pulmonic valvular stenosis (Fig. 5). At this time a superior vena cava right main pulmonary artery shunt was performed. In the postoperative period circulation to the shunt was aided by keeping her sitting upright at all times. Her arterial oxygen saturation was modestly improved and she made great gains in her physical activity (Fig. 6). Two years later the second-stage operation was performed: anastomosis of the tip of the right atrial appendage to the main pulmonary artery (Fig. 7). Inferior vena cava pressure

and flow were increased to help maintain shunt patency by placing the child in a rocking bed in the immediate postoperative period which changed her from a head-up to a head-down position 23 times per minute. Eight months later the atrial septal defect was closed without difficulty, utilizing cardiopulmonary bypass. Her systemic saturation and vena cavae pressures were normal (Fig. 8). The first 24 hours following this operation were uneventful, but then she developed an acute renal shutdown and subsequent hyperkalemia. A cardiac arrest occurred 36 hours postoperatively and resuscitation was unsuccessful.

The complete functional correction of tricuspid valve atresia thus seems to be possible. However, long-term follow-up studies are not available at present, nor are a significant number of cases.

Correction of Ebstein's Malformation

The abnormal position of the tricuspid valve with its displacement into the right ventricle is known as Ebstein's malformation. Correction

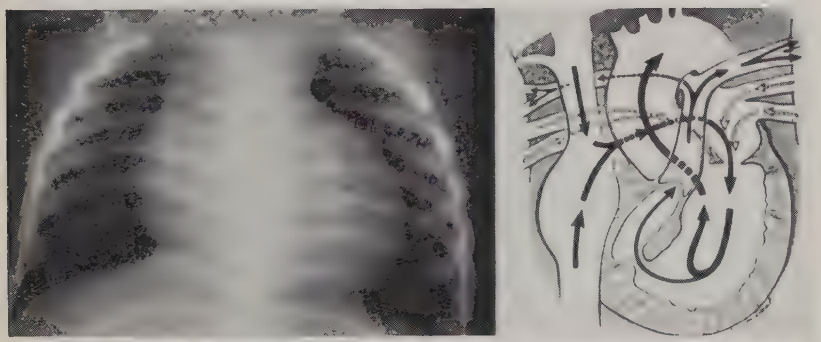

Figure 5. (Tricuspid atresia: Patient S.B. at 15 months). *Left,* Preoperative chest x-ray. *Right,* Diagram of intracardiac blood flow.

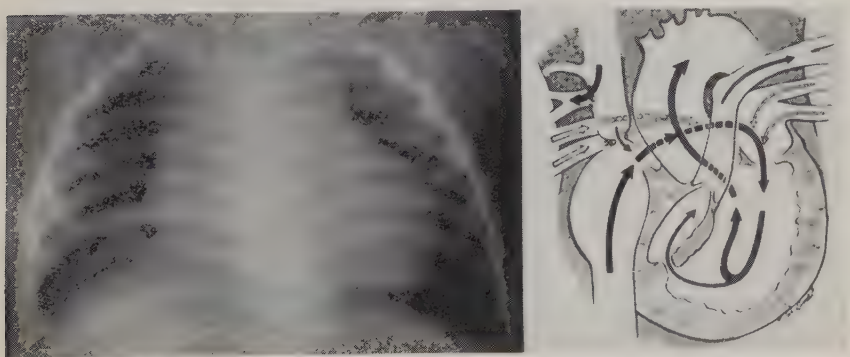

Figure 6. (Patient S.B. at 16 months). One month after superior vena cava to right pulmonary artery (Glenn procedure) anastomosis. Increased vascular markings to right lung are noted. Arterial O_2 saturation increased to 61 per cent.

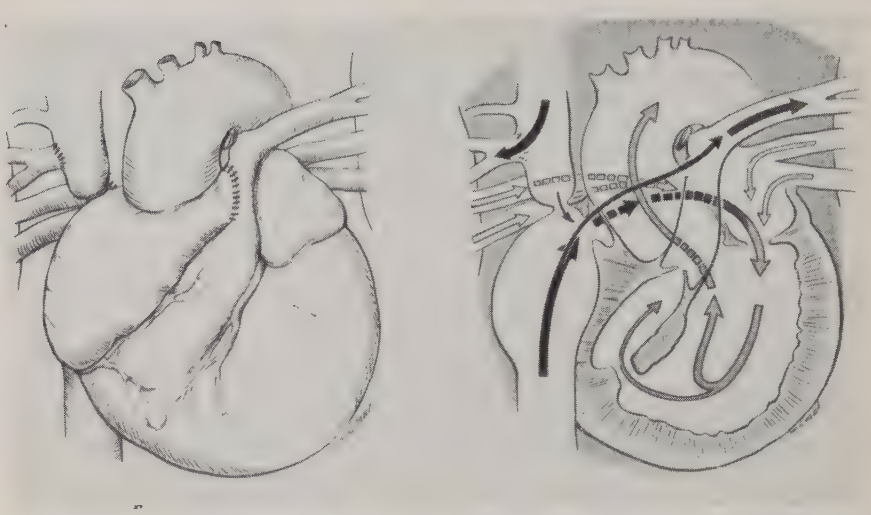

Figure 7. (Patient S.B. at 37 months). Second stage of correction. Right atrial appendage anastomosed to main pulmonary artery. Arterial O_2 saturation increase to 83 per cent.

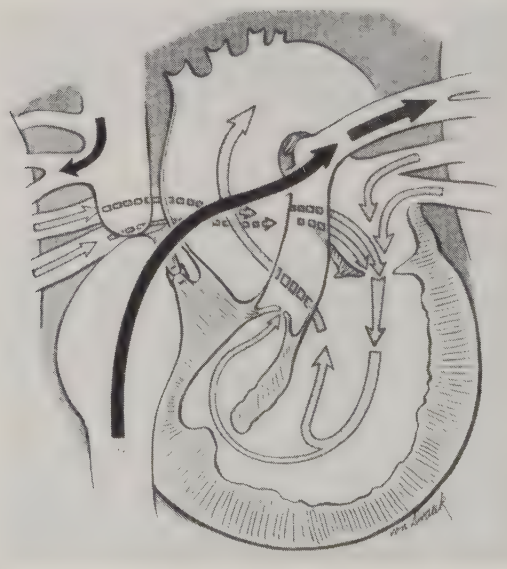

Figure 8. (Patient S.B. at 45 months). Final complete correction with closure of atrial septal defect. Normal arterial O_2 saturation and superior and inferior vena cavae pressures are recorded.

Figure 9. (Ebstein's malformation: Patient G.K.). *Above* (April, 1964), Preoperative chest x-rays. *Below* (September, 1964), Marked decrease in size of the right atrium is evident in the 5 months' postoperative x-rays.

of this defect depends on the condition of the valve leaflets. Hunter and Lillehei[9] first suggested complete correction of this anomaly by transposing the valve leaflets. However, if the valve leaflets are deformed, they must be removed and a prosthetic valve inserted.[1, 13] While the few reported cases indicated good results, the hazard of a prosthesis is present. The presence of an atrialized right ventricle distal to the valve is also of concern throughout life. With this new position distal to the right atrioventricular valve it may not be able to propel blood satisfactorily and right ventricular failure may result.

If a patient with this malformation is fortunate to have a fairly normal valve, it is possible to return the valve and its annulus to the annulus fibrosus. This has been accomplished by Hardy and his associates.[8] Our experience as seen in the following case parallels theirs.

Patient G.K., a 15 year old Greek boy, was admitted to the clinics complaining of dyspnea and fatigue. Evaluation demonstrated cardiomegaly and multiple arrhythmias, particularly sinus tachycardia. An angiogram showed an abnormal right atrium, displaced tricuspid valve, and atrial septal defect. Operation was undertaken with the use of a heart-lung bypass. Exploration identified a small atrial septal defect, atrialization of most of the right ventricular wall and displaced, fairly normal tricuspid valve leaflets. The tricuspid valve annulus was returned to the annulus fibrosus using interrupted 2-0 dacron sutures. This produced a nonfunctioning diverticulum of the atrialized right ventricle, which did not appear to effect function in the postoperative period. The atrial septal defect was closed by direct suture. Recovery has been good. Heart size is normal (Fig. 9) and rate is regular. Angiocardiogram at 8 months showed a small right atrium and right ventricle, absence of tricuspid regurgitation, and some filling of the ventricular diverticulum.

Improvement in patients with Ebstein's malformation rests on a correction of the valve defect. Weinberg et al.[20] have shown that the Glenn procedure may be detrimental in some patients by allowing a reversal of the shunt and continued load on the right ventricle.

SUMMARY

An optimistic note has been sounded by a re-evaluation of older surgical techniques coupled with recent innovations allowing the palliation and frequently the correction of many complex cardiac anomalies in children who were previously considered untreatable.

New operations for pulmonary hypertension associated with intracardiac shunts have been presented in detail along with the correction of complete transposition of the great vessels, tricuspid valve anomalies and pulmonary atresia.

REFERENCES

1. Bahnson, H. T., Bauersfeld, S. R. and Smith, J. W.: Pathological anatomy and surgical correction of Ebstein's anomaly. Circulation (Suppl. I) *31:* 1–3, 1965.
2. Benjamin, R. B., Flom, R. S., MacLean, L. D. and Lewis, F. J.: The gradual closure of interatrial defects. J. Thoracic Surg. *34:* 679, 1957.
3. Bernhard, W. F., Fritelli, G., Task, E. S. and Carr, J. G.: Hyperbaric surgery in infants with congenital cardiac disease. Circulation (Suppl. I) *31:* 91, 1965.
4. Blalock, A. and Hanlon, C. R.: Surgical treatment of complete transposition of the aorta and the pulmonary artery. Surg. Gynec. & Obst. *90:* 1, 1950.
5. Braunwald, N. S. and Morrow, A. G.: The delayed closure of atrial septal defects with perforated prosthesis. Surg. Gynec. & Obst. *116:* 579, 1963.
6. Cassels, D. E.: International Symposium on Heart and Circulation in the Newborn and Infant. New York, Grune & Stratton, 1965.
7. Gilbert, J. W., Callard, G. M. and Barnes, R. W.: Delayed closure of perforated atrial septal prosthesis. J. Thoracic & Cardiovasc. Surg. *50:* 68–73, 1965.
8. Hardy, K. L., May, I. A., Webster, C. A. and Kimball, K. G.: Ebstein's Anomaly: A functional concept and successful definitive repair. J. Thoracic & Cardiovasc. Surg. *48:* 927, 1964.
9. Hunter, S. W. and Lillehei, G. W.: Ebstein's malformation of the tricuspid valve with suggestion of a new form of surgical therapy. Dis. Chest *33:* 297, 1958.
10. Keith, J. D., Rowe, R. D. and Vlad, P.: Heart Disease in Infancy and Childhood. New York, Macmillan Co., 1958, p. 422.
11. Kirklin, J. W. and DuShane, J. W.: Indications for repair of ventricular septal defects. Am. J. Cardiol. *12:* 75, 1963.
12. Kirklin, J. W. and DuShane, J. W.: Repair of ventricular septal defects in infancy. Pediatrics *27:* 961, 1961.
13. Lillehei, C. W. and Gannon, P. G.: Ebstein's malformation of the tricuspid valve, method of surgical correction utilizing a ball-valve prosthesis and delayed closure of atrial septal defect. Circulation (Suppl. I) *31:* 1–9, 1965.
14. Moulder, P. V.: Pulmonary hypertension. Postgrad. Med. *37:* 686, 1965.
15. Muller, W. H., Jr. and Dammann, J. F., Jr.: Treatment of certain congenital malformations of heart by creation of pulmonic stenosis to reduce pulmonary hypertension and excessive pulmonary blood flow. Surg. Gynec. & Obst. *95:* 213, 1952.
16. Mustard, W. T., Keith, J. D., Trusler, G. A., Fowler, R. and Kidd, L.: Surgical management of transposition of the great vessels. J. Thoracic & Cardiovasc. Surg. *48:* 6, 953, 1964.
17. Reis, R. L., Lay, W. A., Jr., Braunwald, N. S. and Morrow, A. G.: Gradual closure of aortopulmonary septal defects. J. Thoracic & Cardiovasc. Surg. *49:* 955, 1965.
18. Senning, A.: Surgical correction of transposition of great vessels. Surgery *45:* 966, 1959.
19. Sloan, H., MacKenzie, J., Morris, J. D., Stern, H. and Sigmann, J.: Open Heart Surgery in Infancy. J. Thoracic & Cardiovasc. Surg. *44:* 459, 1962.
20. Weinberg, M., Jr., Bioff, J. P., Agustsson, M. H., Steiger, Z., Gasul, B. M., Fell, E. H. and Laun, L. L.: Surgical palliation in patients with Ebstein's anomaly and congenital hypoplasia of the right ventricle. J. Thoracic & Cardiovasc. Surg. *40:* 310, 1960.

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Surgery of Acquired Heart Disease

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VALVULAR DISEASE

In 1928 Cutler, Levine and Beck[4, 5] reported the use of a closed transventricular operation for mitral stenosis. There was silence until 1947 when Harken[10] and Bailey[1] separately approached this valvular lesion again. A variety of closed techniques for all types of valvular abnormalities followed through the early 50's. When the extracorporeal circulation techniques were added to allow direct vision, further work was done on the valve directly and soon portions of the valve were being replaced, especially the leaflets of the aortic valve. This accelerating interest soon led to total valve replacement. The present standard, the Starr-Edwards ball valve, was first implanted only five years ago[15] and now there have been almost 25,000 valves delivered by the manufacturer!

Mitral Stenosis

Finger and instrumental dilation continues to be standard procedure for initial and reoperative treatment. There are some who believe that all cases should be approached under direct vision with the use of the pump oxygenator—the strongest argument being that of Kay and Zimmerman;[11] however, they present sequential series and the patients given the closed approach were not operated upon with the use of a dilator. Most surgeons prefer the open technique when considerable calcification of the valve and/or mitral insufficiency are present.

Mortality ranges from 3 to 10 per cent, but is less than 1 per cent in the good candidates. Good results will obtain in approximately 75 per cent, long-term excellent results in 50 to 60 per cent. The incidence of re-stenosis is continuing to rise and the true number will be known better when a plateau is reached. Its reality is emphasized by the occurence of second and third re-stenoses.

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Mitral Valvular Insufficiency

Although two apparent arguments remain as to the surgical therapy: plication-repair vs. prosthetic valve replacement and the type of valve to use, there is a pattern of conformity as follows. For the widely dilated but minimally deformed valve plication of the annulus to an initially overcorrected position is performed. Plication must be carefully performed at the commissural sites with care to preserve the available good valve leaflet substance, and it must be recognized that central loss of substance cannot be overcome. Very rarely will anyone attempt vigorous debridement of calcium deposits, addition of annular substance to a leaflet, and similar procedures—the severely deformed valve will be replaced.

A variety of valves is being used by the developers, but the standard valve is the Starr-Edwards ball valve (*vide infra*).

Mortality statistics as in all cardiac surgery in its evolutionary and developmental stages varies primarily with the type of patient and secondarily with many other factors, but generally it is 15 to 20 per cent; however, with selected cases this has been less than 10 per cent for experienced surgical groups. There remains a definite late mortality related to the prosthesis of around 5 per cent, usually due to embolism.

Clinical improvement in the early stages has been excellent, but it is too early for definitive assessment of the results in any way comparable to mitral valvulotomy. There is also a continuing morbidity from thromboembolism.

Aortic Valvular Disease

In the late 50's and early 60's a great effort was put into techniques for repair, debridement, sculpturing, etc., on the deformed aortic valve and then on single to multiple leaflet replacement. The former methods have almost been abandoned as the late follow-up has revealed an astonishing recurrence of the malformation.[13] (It reminds one of the not uncommon marked increased calcification seen in recurrent mitral stenosis.) Leaflet replacement has been abandoned completely,[12] as use of the leaflets in perfect coaptation led to early insufficiency, overcorrection led to late irregularity with sudden onset of insufficiency; even those in reasonable position suffer in time from loss of mobility and cracking with recurrent stenosis and insufficiency. Many patients with such problems have been subjected to reoperation with the insertion of a prosthetic valve.

Again the Starr ball valve is the standard.

The mortality rates are slightly higher than in the mitral valve replacement and there is also an embolism problem. Recently ball deformity and even extrusion have been described in 17 cases (note none in the mitral implantations), and at the moment this uncommon complication must be

ascribed to technical placement and sizing difficulties, which emphasizes the need for care and skill in the use of these valves.

Prosthetic Valves

As mentioned, the Starr-Edwards ball valve is the standard, but even it has undergone changes. A few lenticular or disc valves have been developed in an attempt to decrease the cage size. Resistance to opening and loss of orifice area to the necessary base common to these types of valves would be greatly obviated by a leaflet valve, tricuspid in the aortic site as is the natural valve, but great efforts have failed to produce an acceptable one. The major problem associated with all valvular prostheses and clearly with the Starr valve because of the large experience with it is early and late thrombosis with embolization. Two approaches in valve design have been made to overcome this, one to increase surface resistance to clot formation and the other to induce incorporation of normal surfacing endothelium on as much as possible of the prosthesis. Albert Starr is implanting mitral valves in which the entire base is cloth-covered to promote endothelialization, and Davila is working with a valve in which the entire contact base will be incorporated in tissue and the valve mechanism will float free.

Another plastic valve for overcoming these problems in clinical trials is that of Vincent Gott.[9] He developed the graphite-benzalkonium-heparin surface for coagulation resistance and it is incorporated in all parts of the valve except the suturing base and the leaflets. For the latter a hinged flap of silicone-covered dacron cloth is employed, seeking low resistance and a large orifice to total size relationship.

Barratt-Boyes[2] in New Zealand and Ross[6, 14] in England have been replacing the aortic valve with homografts and have a startlingly good follow-up showing maintenance of valve function. This valve should not have the thromboembolic problems of the prostheses.

Thus it can be said that the Starr valve is an excellent one, it is in general use and has been significant in contributing to the healing and rehabilitation of thousands of patients who represent our prime concern. Nevertheless, Albert Starr is the first to recognize that better valves will appear (and that the best may be another Starr).

HEART BLOCK

Atrioventricular dissociation associated with chronic and incapacitating bradycardia or with episodic asystole (Stokes-Adams) and its cerebral sequelae can be treated effectively with implantable pacing electronic units, with many years of function obtainable before replacement is necessary.[3, 16]

The basic concept in technique is standard. As soon as the patient is

seen in trouble or when permanent pacing is planned, a bipolar catheter is inserted percutaneously in the heart via a peripheral vein. The heart rate is then set at a normal level and a period of hours or days is allowed to get the patient into appropriate condition for elective placement of a pacemaker.

At the present time our clinical group and others are using a similarly placed transjugular venous catheter with subcutaneous insertion of the pacemaker unit. The more common plan is to implant electrodes on the left ventricular myocardium and place the pacemaker in the lateral chest wall, in the abdominal wall (subcutaneous or subfascial), or even in the posterior thoracic cavity.

The pacemaker units have attained considerable reliability, but the stimulating electrodes have not done as well. Methods for obtaining longer or even permanent energy life for the units are being sought. Even though replacement is technically simple, it is a hazard and costly.

One unit undergoing evaluation in considerable numbers uses an atrial signal to induce ventricular excitation. This is theoretically intriguing, and desirable for the younger and otherwise healthy individual.

CORONARY ARTERY DISEASE

The new operations for coronary artery disease are not spectacular as yet because of their limited application and because of the continued difficulties in selection and evaluation of patients.

Application of direct vascular techniques to the coronary arteries has been done by Longmire and colleagues[7] for multiple and long segment obstructions with limited but occasionally real success. Until recently Effler has performed arteriotomy and patch grafting on short segment, large (proximal) vessel obstruction with marked surgical success. Rivival of the Vineberg operation, implantation of the internal mammary artery into the myocardium, can be considered new and is undergoing use and evaluation in many areas.[8]

For these procedures definitive evaluation requires good coronary arteriography and even with this one must recognize that many patients have obstructive disease as severe as that present in the surgical candidate and their performance can be normal. Although relief of obstruction and refilling of distal vessels in the arteriogram are reassuring and provide supportive data, one would like a direct assessment of myocardial performance in addition.

It is probable that small vessel surgery will soon be improved to a point of general usefulness and this should afford the opportunity to treat large numbers of patients with severe coronary artery disease in a suitable random fashion to back up these initially good results.

REFERENCES

1. Bailey, C. P.: Surgical treatment of mitral stenosis (mitral commissurotomy). Dis. Chest *15:* 377, 1949.
2. Barratt-Boyes, B. G.: Presentation at American Heart Association Meeting, October, 1965, Bal Harbour, Florida.
3. Chardack, W., Gage, A. and Greatbatch, W.: A transistorized, self-contained, implantable pacemaker for the long-term correction of complete heart block. Surgery *48:* 643, 1960.
4. Cutler, E. C. and Beck, C. S.: Present status of the surgical procedures in chronic valvular disease of the heart. Arch. Surg. *18:* 402, 1929.
5. Cutler, E. C. and Levine, S.: Cardiotomy and valvulotomy for mitral stenosis: Observations and clinical notes concerning an operated case with recovery. Boston Med. & Surg. J. *188:* 1023, 1923.
6. Davies, H., Lessof, M. H., Roberts, C. I. and Ross, D. N.: Homograft replacement of the aortic valve. Follow-up studies in twelve patients. Lancet *1:* 926, 1965.
7. Dilley, R. B., Cannon, J. A., Kattus, A. A., MacAlpin, R. N. and Longmire, W. P.: Treatment of coronary occlusive disease by endarterectomy. J. Thor. & Cardiov. Surg. *50:* 511, 1965.
8. Effler, D. B., Sones, F. M , Groves, L. K. and Suarez, E.: Myocardial revascularization by Vineberg's internal mammary artery implant. J. Thor. & Cardiov. Surg. *50:* 527, 1965.
9. Gott, V. L., Daggett, R. L., Whiffen, J. D., Koepke, D. E., Rowe, G. G. and Young, W. P.: A hinged-leaflet valve for total replacement of the human aortic valve. J. Thor. & Cardiov. Surg. *48:* 713, 1964.
10. Harken, D. E., Ellis, L. B., Ware, P. F. and Norman, L. R.: Surgical treatment of mitral stenosis. New England J. Med. *239:* 801, 1948.
11. Kay, E. B., Rodriguez, P., Haghighi, D., Suzuki, A. and Zimmerman, H. A.: Mitral stenosis. Comparative analyses of postoperative results following the closed and open approach. Am. J. Cardiol. *14:* 139, 1964.
12. Larson, R. E. and Kirklin, J. W.: Early and late results of partial and total replacement of the aortic valve with individual teflon cusps. J. Thor. & Cardiov. Surg. *47:* 720, 1964.
13. Mulder, D. G., Kattus, A. A., Fonkalsrud, E. W. and Longmire, W. P.: Surgical treatment of acquired aortic stenosis. J. Thor. & Cardiov. Surg. *46:* 468, 1963.
14. Ross, D. N.: Homotransplantation of the aortic valve in the subcoronary position. J. Thor. & Cardiov. Surg. *47:* 713, 1964.
15. Starr, A. and Edwards, M. L.: Mitral replacement: Clinical experience with a ball valve prosthesis. Ann. Surg. *154:* 726, 1961.
16. Zoll, P. M., Frank, H. A. and Linenthal, A. J.: Four-year experience with an implanted cardiac pacemaker. Ann. Surg. *160:* 351, 1964.

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Vena Cava Operations for Prevention of Pulmonary Embolism

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It is a fact of medical practice that vena cava ligation prevents recurrent pulmonary embolization; nevertheless, this excellent operation is poorly accepted by the general medical population. Fear, real or imagined, exists that stasis sequelae develop following this procedure, and anxiety regarding known and unknown effects of such a seemingly ominous surgical attack has restricted its application. Indications for cava ligation are therefore quite stringent; for example, some busy vascular services report as few as 12 or 15 such operations over a long span of years.[7, 22]

It is possible that some patients with pulmonary embolization are being denied adequate surgical protection against recurrence. If this is true, cava ligation has failed its chief assignment—the protection of as many patients as possible against repeated pulmonary emboli.

The ground swell of perhaps unjustified dissatisfaction with cava ligation has caused development of new operations. All of these are designed to protect patients from pulmonary infarction yet preserve patency of the vena cava. Historically, the first of these advocated a temporary catgut ligature of the cava,[6, 11] but this was quickly abandoned. Almost simultaneously a suggestion was made that a metal clip could be used to narrow the cava, thus entrapping clots but allowing continued blood flow.[14] The metal clip was discarded in favor of Teflon when this material was shown to be superior and this modified device is in current clinical

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use.[12] DeWeese suggested a harp-string grid, or filter, which could be constructed within the cava[8] and the results with this operation are impressive.[9] Later, Spencer noted that compartmentation of the cava, or plication, could be effective in halting emboli while preserving cava blood flow.[20] Some dissatisfaction with this operation has been registered from several sources.[3, 6]

INDICATIONS

In the past, when femoral vein ligations were utilized in management of patients with known or suspected thromboembolic disease, prophylactic operations were performed. In many instances bilateral femoral ligations were done when calf clotting was diagnosed even before pulmonary embolization. Similarly, in high-risk subjects, such as aged patients with femoral neck fractures, prophylactic femoral venous ligations were performed even before deep thrombophlebitis occurred.

Vena cava ligation is not commonly done for prophylaxis. Well defined and conservative indications for this operation are recognized to be as follows:

1. A pulmonary embolus occurring in a patient in whom anticoagulants are contraindicated.
2. A pulmonary embolus occurring in a patient under full and adequate anticoagulant therapy.
3. Need for acute cessation of anticoagulant therapy in a patient with a prior pulmonary embolus.
4. Chronic multiple pulmonary emboli causing pulmonary hypertension.
5. Septic pelvic thrombophlebitis.

These indications imply that the first line of defense in treatment of pulmonary embolization is anticoagulant therapy. This is true and, additionally, the most effective anticoagulant available for this use is heparin. The heparin is given intravenously when either deep venous thrombosis or a pulmonary embolus is suspected. It is continued for 10 to 12 days, at which time an oral prothrombin depressant anticoagulant is substituted. Treatment is continued with the latter for a prolonged period dependent upon the patient's underlying disease process, prior history of phlebitis, and response to therapy. A minimum time for anticoagulant therapy following pulmonary embolization is six weeks. Anticoagulation with heparin requires the clotting time to be prolonged to at least twice normal values.

After a single nonfatal herald pulmonary embolus the risk of a recurrent and possibly fatal embolus is so great that immediate therapy is

mandatory. If anticoagulants cannot be given, as in patients with head injury, extensive recent surgical procedures, or recent gastrointestinal hemorrhage, a vena cava control operation must be done. Similarly after a herald embolus, if anticoagulants must be discontinued for any reason such as difficulty of adequate control or frank hemorrhage, the cava must be controlled. This important indication is easily forgotten and fatal recurrent embolus has occurred as heparin was being stopped in a patient with gastrointestinal hemorrhage following a pulmonary infarct.

The other indications are self-explanatory and are well accepted. An embolus occurring during adequate anticoagulant therapy announces failure of such treatment. It is remarkable in reviewing the records of patients being treated with anticoagulants for pulmonary embolization how often second and third emboli can be diagnosed from the retrospective vantage point. This emphasizes the difficulty of accurate diagnosis of this condition. Our current experience suggests that plasma enzyme determinations—in particular a rising lactic dehydrogenase while the glutamic oxaloacetic transaminase remains normal—is helpful in one-third of patients. The pulmonary vascular radioisotope scan is much more reliable and when supplemented with an inhalatory scan is totally accurate. These methods supplement and are far superior to the other classically cited diagnostic methods.

TECHNIQUE

Surgical exposure of the vena cava is similar regardless of the operation to be done on this structure. Usually an extraperitoneal approach is employed and this is entirely satisfactory. Sometimes, especially when the procedure is used in connection with intra-abdominal surgery, or when the postpartum state dictates the need for concomitant ovarian vein ligation, the transperitoneal approach is utilized.

Local anesthesia for cava surgery is unsatisfactory. Spinal anesthesia is usually contraindicated because of the systemic anticoagulation used preoperatively to treat prior phlebitis or pulmonary embolism. Therefore, general anesthesia is usually given despite the fact that severe cardiopulmonary insufficiency is regularly present in these patients.

In performing the extraperitoneal operation, the exposure is similar to that employed for sympathectomy (Fig. 1). A skin incision is made from the tip of the eleventh rib toward the umbilicus. This halts at the lateral edge of the rectus sheath. As the incision is deepened through the subcutaneous fat, the medial and lateral wound edges are undermined to allow splitting of the fibers of the subjacent external oblique muscle. When this muscle is incised from the rib margin to the rectus

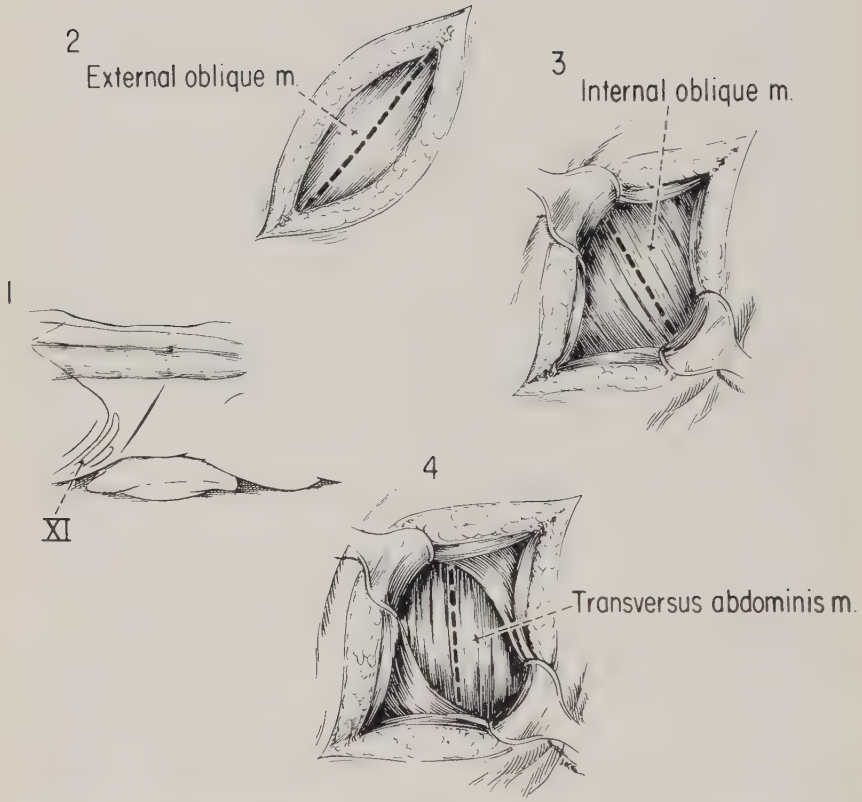

Figure 1. Vena cava exposure by a retroperitoneal muscle-splitting incision.

1. The skin incision angles from near the tip of the eleventh rib toward the umbilicus. Little is gained by extending the incision over the rectus muscle.

2. The external oblique muscle is incised from the rib margin to the edge of the rectus sheath.

3. After wide mobilization of the external oblique muscle, the internal oblique is incised from the iliac crest to the edge of the rectus muscle.

4. Access to the retroperitoneal space is gained by an incision in the transversus abdominis muscle.

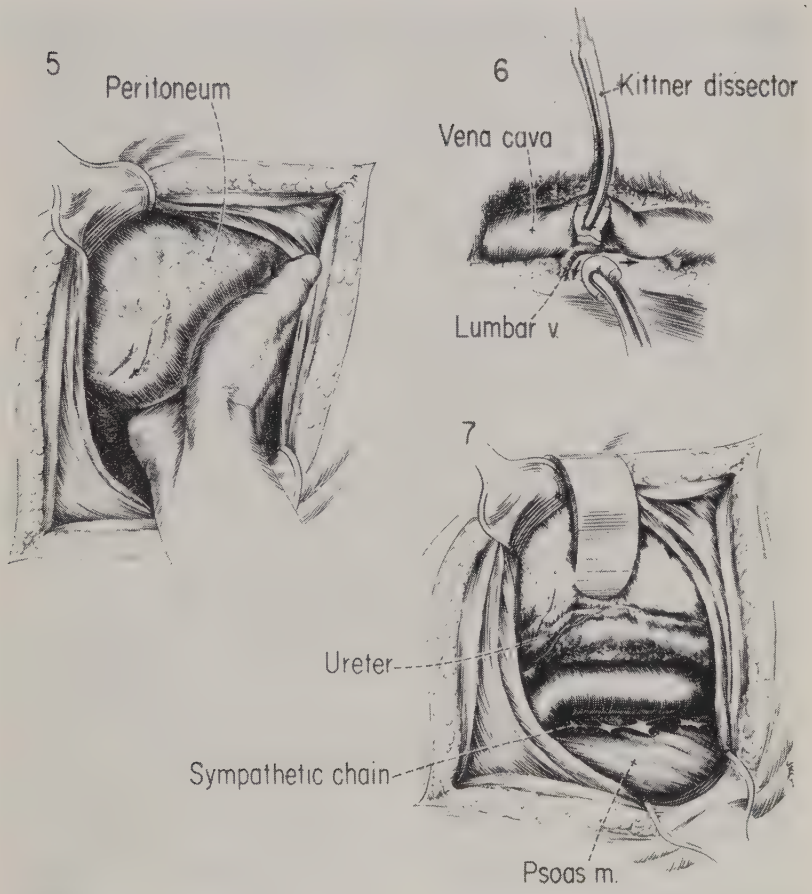

Figure 2. Mobilization of the peritoneum and vena cava for definitive cava control to prevent recurrent pulmonary embolism.

5. As the peritoneum is mobilized medially, care is exerted to prevent entering the retropsoas space.

6. Careful direct vision dissection using cotton-tipped sponges allows wide exposure of the cava without damage to the fragile lumbar veins.

7. A 6-cm. exposure of the vena cava is sufficient for performance of the definitive control procedure.

sheath, its undersurface is widely freed from the internal oblique. Later adequate deep exposure is dependent upon early wide mobilization of each anatomical layer.

Careful splitting of the fibers of the internal oblique muscle prevents inadvertent entering of the peritoneal cavity. Protection of the peritoneum is offered by the transversus abdominis muscle and properitoneal fat. The iliac crest and rectus sheath define the margins of the internal oblique muscle incision.

An attenuated transversus abdominis muscle is usually encountered at this midabdominal level. Its fibers are initially separated laterally in order to avoid the underlying peritoneum. This is adherent to the transversus abdominis muscle medially. The muscle split is then enlarged medially to the rectus sheath and laterally as far as exposure allows.

The retroperitoneal space is next exposed. Now, adequate muscle relaxation is mandatory. Exposure of the cava will be hazardous if coughing and straining are allowed to occur.

A hand is inserted into the incision and the peritoneum is mobilized widely superiorly and inferiorly (Fig. 2). The iliac fossa serves as a natural guide to this blind development of the peritoneum. A conscious effort is made to avoid the retropsoas space since it is tempting to drop behind the psoas as the peritoneum is being mobilized medially. The blunt areolar tissue dissection is complete when the spine and sympathetic chain can be palpated easily in the depths of the wound.

At this point, placement of retractors exposes the cava. A broad retractor is placed anteriorly and held strongly in a medial direction against the peritoneum. Narrow curved retractors are then placed to retract pericaval fat superiorly and inferiorly. These retractors are essential to a continuous unobstructed view deep in the wound. They are held immobile. Adjustment of the retractors is made only by the surgeon.

The first assistant now stands next to the surgeon in order to gain a better view of the deep structures.

Axial rotation of the operating table, dropping the left side, will allow the assistants to see the cava while they retract. The vena cava and iliac veins should be palpated to ascertain presence or absence of intraluminal clot.

Easy dissection of the vena cava is dependent upon development of the proper adventitial plane. A lipo-areolar layer envelops the vessel and must be split for 6 to 8 cm. on its lateral surface. When this is done, a cotton-tipped dissector can be used to free the entire anterior and posterior surface of the vessel.

Often one small cotton dissecting sponge can be used to hold the cava while a second wipes away the areolar investing tissues so that the lumbar veins can be identified. These thin-walled vessels tear easily at

their caval attachment and bleed ferociously. Direct-vision dissection of the vena cava insures against accidental tearing of tributary veins. A 6-cm. length of cava is exposed and then a curved clamp is passed medially and posteriorly to encircle this vessel for positive control. This clamp catches an atraumatic cotton or rubber sling which aids further mobilization by allowing appropriate traction. At no time is blind dissection used. The clamp tip is watched at all times while passing behind the cava so that lumbar veins can be avoided. Even the medial caval attachments near the aorta are divided under direct vision.

With the cava mobilized to the extent described, any of the following procedures can be performed expeditiously. It should be noted that much less exposure is needed for simple ligation. Similarly, less dissection is needed for application of a Teflon clip[13] or placement of Spencer's new cava clamp.[21]

The above-detailed surgical exposure of the cava is used by many surgeons and it is well described by Madden.[11] An interesting muscle-cutting variant, which we have not used, has been suggested by Veith et al.[23]

Definitive Operations

If cava *ligation* is elected, a ¼-inch cotton umbilical tape is passed once around and tied snugly against the cava (Fig. 3). While this is our preference, some authors have suggested two ligatures of 2-0 silk or even No. 2 silk. Catgut is no longer advocated. Ligation is by far the simplest cava operation. It can be accomplished swiftly if the surgical procedure must be terminated quickly.

If a cava *clip* is to be used, it is applied perpendicular to the cava. The clips available commercially vary but their techniques of application are alike in that the clip is placed to flatten the cava transversly (Fig. 3). Moretz' clip is 4 cm. long and has a 4-mm. gap. Other varieties including a serrated device which compartments the cava are being marketed.

In the performance of the cava *plication* procedure, the cava walls are usually approximated with two parallel occlusive atraumatic vascular clamps. A single row of mattress sutures is then placed. These are carefully spaced 5 mm. apart and an adequate bite of vessel wall is taken to insure against tearing through (Fig. 3). Three or four sutures of 4-0 or 5-0 nonabsorbable suture material are placed and tied. Our preference in suture material has been Teflon-impregnated dacron* or dacron alone, but Spencer has used silk in his cases. Care is taken so that the parallel channels thus created are at least 4 mm. in diameter.

The *filter* procedure of DeWeese differs from the plication operation mainly in the amount of tension applied to the cava wall. Also, in placing

* Supplied as Tevdek by Deknatel, Inc.

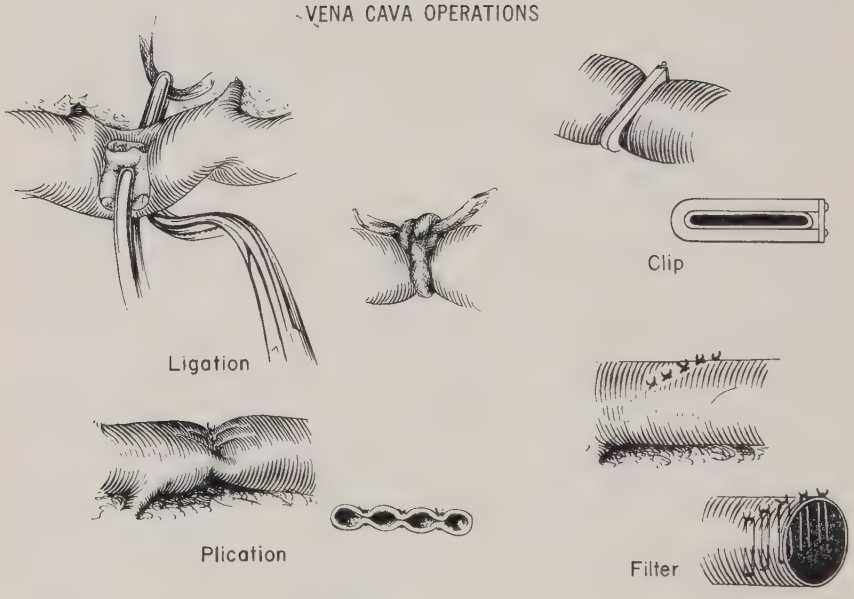

Figure 3. Vena cava operations.

Ligation. Cava ligation is accomplished by passing a narrow cotton tape once around and knotting it snugly against the cava.

Clip. Application of the Teflon clip flattens the vena cava transversely thus narrowing its lumen.

Plication. A single row of mattress sutures are placed and tied to convert the vena cava into a series of parallel channels.

Filter. In performing the vena cava filter operation, the mattress sutures are tied so as to leave a screen within the cava lumen.

the mattress sutures for the filter, the cava is compressed gently only with tissue forceps. Atraumatic clamps are not used. The suture-carrying needle is straightened so that it penetrates the cava in a direct antero-posterior plane both in the initial suture and in the return. Five or six separate sutures are placed in an oblique line so that entrapped clots are directed toward the lateral cava wall (Fig. 3). Thus 10 or 12 threads are placed within the cava lumen. Once again, 5-0 Teflon-coated dacron is preferred. In this instance the choice of suture material is somewhat critical. Absence of tissue reaction is essential if cava thrombosis is to be avoided. In time, the suture will become endothelialized.

DeWeese performs the cava filter operation through a transperitoneal transverse supraumbilical incision and uses a continuous suture rather than five or six interrupted stitches. The contnuous suture allows adjustment of tension so that the cava is not too severely indented. When interrupted sutures are used, some care is required in adjusting the tension of each knot. No attempt is made to narrow the cava.

RESULTS

A detailed analysis of the results of our experience with vena cava plication has been reported.[3] Similarly, a comparison of cava ligation, plication and the filter operation is now complete.[4] Reviews of cava ligation experiences are available for comparison of these procedures.[10, 16, 19]

Some general conclusions can be drawn from the published records of these operations. Dale's classic review of cava ligation experience shows that the complication rate varies widely in the published series.[5] He concludes that edema occurs frequently after cava ligation. Persistent, troublesome leg edema develops in one-third of patients. This has led to permanent disability in an occasional patient. In most patients control of complications has been possible but postligation edema is frequent enough to have been the concern of many investigators. In general, its occurrence has been blamed upon preoperative phlebitis rather than on cava ligation per se.

There has been much criticism leveled against cava ligation but it is conceded that the operation is effective protection against recurrence of pulmonary infarction. While occasional postligation emboli have been reported, the infrequency of their occurrence testifies to the efficacy of the operation.[13] It is important to recognize this fact, for all of the new operations must match this basic standard of success if they are to achieve general acceptance.

Vena cava plication is apparently effective in protecting patients against recurrent pulmonary emboli. Spencer reports only one patient

in 39 who had a postplication pulmonary embolus.[21] None of our ten surviving patients had a recurrent embolus.[3] Nevertheless, it is apparent that a postplication embolus can occur as shown by the well documented case described by Dale.[6] This patient had a fatal postoperative pulmonary embolus and at autopsy was found to have a clot protruding through the plication site.

The cava plication has been shown to trap clots effectively. These thrombi have been demonstrated angiographically as well as at autopsy. In addition, experimental studies have confirmed this fact.[1]

Persistent patency of the plication site has been questioned. Some difficulty arises in accurately determining patency of the vena cava. It has now been well demonstrated that clinical signs of venous pressure and presence or absence of edema are fallacious. Only contrast visualization of the cava by x-ray is exact. Our own group of patients with plication demonstrated this to be a fact. In this group, only three cavae of nine studied with postoperative cavagrams were found to be patent. Only three of the six patients with occluded cavae were symptomatic. In contrast, in Spencer's large group of 39 cases, 23 patients had postoperative cavagrams and in 20 of these the cava was found to be patent. Furthermore, Spencer showed that one vena cava which occluded at the plication site recanalized and became patent within 12 months. This is an impressive and important finding. The restudy of our own patients up to three years after plication has failed to show any recanalization.

The filter procedure is also effective in preventing recurrent emboli. In our 12 cases, no recurrent pulmonary embolus has been diagnosed. In several instances, when postoperative chest pain occurred, plasma enzymes, radioisotope scan, and clinical findings did not confirm the presence of a suspected embolus. The patients who experienced recurrent chest pain usually manifested this at the site of a previous pulmonary infarct. This phenomenon was also seen in one patient with a cava ligation.

DeWeese also shows the effectiveness of the filter operation but reports the course of one patient who apparently sustained a fatal pulmonary embolus after a cava filter was placed. This report necessarily tempers enthusiasm for the procedure. Trapping of emboli by the filter has been demonstrated by cavagrams and by autopsy studies.

None of our 12 patients have had cava thrombosis following the filter operation. All but one have had postoperative cavagrams. James DeWeese has reported venous occlusion in two of three patients.[1] Marion DeWeese's larger series indicates that only one of 24 patients has had increased disability following the filter operation but that only five patients had postoperative cavagrams.[9]

Moretz describes a large experience with the partially occluding clip. In 43 patients, three sustained a recurrent pulmonary embolus.

This would imply that this operation is less successful in achieving its objective than the others. He has performed cavagrams in 12 patients and has found partial or full patency in only eight. He adds the most important note that the patients with cava occlusion have developed phlebitis in previously normal extremities while those with open cavae have had no new phlebitis in previously normal extremities.[15] The implications of this observation can be applied to all vena cava operations. That is, that cava occlusion per se may cause sufficient stasis to initiate distal venous occlusion.

In comparing patients with the various operations, especially in comparing cava ligation with the new cava patency procedures, it is pertinent to contrast patients with an open cava with those whose cava is occluded. When this is done, it is clear that those with a closed cava have more edema than patients with an open cava. This is true whether the cava is occluded by ligature, by thrombosis, or by entrapment of emboli. This was found in the present group of 34 cases, in Spencer's plication group, in DeWeese's filter group and in Moretz' clip series.[4, 15, 21] It becomes increasingly apparent that the closed cava leads to more severe edema, requires closer attention to postoperative elastic bandaging of the legs, and predisposes the affected patients to development of new phlebitis in previously nonaffected extremities.

It is of more than incidental interest that the cava thrombosis which may develop following the plication or filter might recanalize and allow later cava patency while retaining the protection against recurrent emboli.

If these observations are confirmed by a larger experience, the doctrine that postligation venous stasis is caused by preoperative phlebitis will have to be discarded. It may be that cava occlusion from whatever cause leads to venous stasis which predisposes patients to distal phlebitis. This recurrent phlebitis may be the cause of the severe edema and persistent and occasionally disabling stasis sequelae that is noted in many reports on cava ligation.

COMMENT

The importance of pulmonary embolization in hospitalized patients cannot be overstressed. The incidence of pulmonary emboli found in consecutive autopsies is uniformly in the range of 12 to 14 per cent. While this indicates the frequency of this entity, it is conceded that errors in clinical diagnosis are made in one-half of these cases.

Analysis of large groups of case records reveals that, if a patient sustains a single herald pulmonary embolus, he has a 43 per cent chance of developing a new, clinically recognizable thrombosis. In addition, he

has a 30 per cent chance of sustaining another pulmonary embolus and an 18 per cent chance of a fatal embolus.[2]

These facts point up the importance of effective treatment of the thrombotic episode and of the thrombosis which inevitably attends pulmonary embolization. Effective anticoagulant therapy with heparin is the first line of such defense. Use of this agent decreases the chance for a recurrent embolus to 10 to 15 per cent and the fatality rate by a similar figure.

As experience is gained with the new operations described above, it may be that they will prove even more effective than heparin and may even be associated with less morbidity. They may provide adequate protection against recurrent emboli and produce no additional stasis sequelae. Therefore, it is possible that the indications attending their application will be much less strict than those of vena cava ligation and that earlier protection will be offered to a greater number of patients.

SUMMARY

New vena cava operations allowing effective prevention of recurrent pulmonary emboli are described. These include cava plication, filtering, and clip application. The indications, technique of performance, and results of such surgery are discussed.

REFERENCES

1. Adams, J. T. and DeWeese, J. A.: Experimental and clinical evaluation of partial vein interruption in the prevention of pulmonary emboli. Surgery *57:* 82, 1965.
2. Allen, E. V., Barker, N. W., and Hines, E. A.: Peripheral Vascular Diseases. 3rd Ed. Philadelphia, W. B. Saunders Co., 1962.
3. Bergan, J. J., Kaupp, H. A. and Trippel, O. H.: Critical evaluation of vena cava plication. Arch. Surg. *88:* 1016, 1964.
4. Bergan, J. J., Kinnaird, O., Koons, K. and Trippel, O. H.: Critical comparison of vena cava ligation, plication and filter operations in prevention of pulmonary embolism. Arch. Surg. (In press.)
5. Dale, W. A.: Ligation of the vena cava for thromboembolism. Surgery *43:* 24, 1958.
6. Dale, W. A.: Discussion of paper by Spencer et al.[21]
7. deTakats, G.: Discussion of paper by Spencer et al.[21]
8. DeWeese, M. S. and Hunter, D. C., Jr.: A vena cava filter for the prevention of pulmonary emboli. Bull. Soc. Internat. Chir. *17:* 17, 1958.
9. DeWeese, M. S. and Hunter, D. C., Jr.: A vena cava filter for the prevention of pulmonary embolism. Arch. Surg. *86:* 852, 1963.
10. Krause, R. J., Cranley, J. J., Hallaba, M. A. S., Strasser, E. S. and Hafner, C. D.: Cava ligation in thromboembolic disease. Arch. Surg. *87:* 184, 1963.
11. Madden, J. L.: Ligation of the inferior vena cava. Ann. Surg. *140:* 200, 1954.
12. Miles, R. M.: The prevention of pulmonary embolism by the use of a plastic vena caval clip. (Presented at the Southern Surgical Association Meeting, December 1964.) Cited by Spencer et al.[21]

13. Moretz, W. H., Rhode, C. M. and Sheperd, M. H.: Prevention of pulmonary emboli by partial occlusion of the inferior vena cava. Am. Surgeon *25:* 617, 1959.
14. Moretz, W. H., Naisbitt, P. F. and Stevenson, G. P.: Experimental studies on temporary occlusion of the inferior vena cava. Surgery *36:* 384, 1954.
15. Moretz, W. H.: Discussion of Spencer et al.[21]
16. Mozes, M., Adar, R., Bogokowsky, H. and Agmon, M.: Vein ligation in treatment of pulmonary embolism. Surgery *55:* 621, 1964.
17. Pualwan, F., Jones, G. E., Bruny, S. J. and Dale, W. A.: Effects of ligation of the inferior vena cava with absorbable suture. S. Forum *5:* 179, 1954.
18. Schauble, J. F., Stickel, D. L. and Anlyan, W. G.: Vena cava ligation for thromboembolic disease. Arch. Surg. *84:* 17, 1962.
19. Shea, P. C. and Robertson, R. L.: Late sequelae of inferior vena cava ligation. Surg. Gynec. & Obst. *93:* 153, 1951.
20. Spencer, F. C.: Experimental evaluation of partitioning of the inferior vena cava to prevent pulmonary embolism. S. Forum *10:* 680, 1960.
21. Spencer, F. C., Jude, J., Rienhoff, W. F. and Stonesifer, G.: Plication of the vena cava for pulmonary embolism. Ann. Surg. *161:* 788, 1965.
22. Szilagyi, D. E.: Discussion of paper by DeWeese and Hunter.[9]
23. Veith, F. J., Marshall, D. C. and Crane, C.: A technique for ligation of the inferior vena cava. Surg. Gynec. & Obst. *119:* 109, 1964.

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Surgical Uses of the Hyperbaric Oxygen Chamber

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In response to the persevering efforts of Boerema of Amsterdam, there has developed in various clinics throughout the world a resurgence of interest in the use of the hyperbaric chamber. Boerema began to use hyperbaric oxygen as a therapeutic agent as early as 1956. Stimulated by his publications in 1961,[8, 11] the clinical world began to take notice of this new approach to an old method of therapy. Actually, there have been numerous brief episodes of clinical interest in the hyperbaric chamber during the past 100 years,[14] but none which compares with the current attention given to it.

The invention of the caisson in France in 1841 to better enable workers to build underwater structures marks the beginning of interest in this device. Subsequently, higher than atmospheric pressures were used by clinicians for reduction of incarcerated hernia (Bert[5]), improvement of nitrous oxide anesthesia (Fontaine in 1879), and treatment of

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ileus (Wangensteen[25]). They were also used in certain European cities during the last century in the treatment of respiratory diseases and various other ailments. Yet the first true surgical operation within a hyperbaric chamber was probably not performed until 1956, when Boerema and his co-workers began their investigations. Many medical centers have now built hyperbaric chambers for clinical research. Data accumulating from these suggest that hyperbaric oxygenation is definitely limited in its clinical applications, though at times it can be lifesaving, and as an investigative tool it has great potentiality.

Hyperbaric oxygenation is considered of unquestioned value in the treatment of clostridial myositis and carbon monoxide poisoning. Its application in numerous other conditions, however, is under continual study, and any conclusions must await the accumulation of more data.

Potential areas of importance of hyperbaric oxygen in surgery are: protection during induced circulatory arrest, procurement of cadaver donor organs for homotransplantation, acute arterial insufficiency due to trauma or frostbite, hypovolemic shock, and laboratory research (e.g., surgically induced coronary occlusion).

PHYSIOLOGIC CONSIDERATIONS

Hyperbaric oxygenation is simple in concept. The patient is placed in a steel or lucite pressure chamber and subjected to a significantly elevated pressure while breathing 100 per cent oxygen either through a tightly fitting mask, or via an endotracheal tube if under anesthesia. In this environment inhaled oxygen becomes dissolved in the plasma in remarkably high concentrations (nearly 20 times normal at 3 atmospheres of pressure). A steep gradient is thus established between the oxygen-carrying plasma and the tissue fluids of any ischemic organ reached by the circulation, and theoretically the transfer of oxygen to that organ should be enhanced thereby. Such transfer does not always occur, however, because of the tendency of hyperbaric oxygen to produce a marked vasoconstriction.

At normal atmospheric pressure, the partial pressure of oxygen in the inspired air is about 150 mm. Hg. Because of the mixing of the inspired gas mixture with the gas mixture in the dead space, the partial pressure of oxygen arriving in the alveoli is only slightly more than 100 mm. Hg. Arterial blood carries oxygen at a pressure of 100 mm. Hg. At this pressure, the hemoglobin carries 19 to 20 ml. (per 100 ml. of blood) of oxygen, whereas only about 0.3 ml. of oxygen is carried in 100 ml. of plasma. When the oxygen content of the inspired gas mixture is increased, the oxygen saturation of hemoglobin is not appreciably altered; however,

the amount of dissolved oxygen in plasma increases according to Henry's law. If the inspired gas mixture is replaced with pure oxygen, the oxygen in physical solution is raised to about 2.00 ml./100 ml. (at a partial pressure of about 700 mm. Hg), at sea-level pressure. If 100 per cent oxygen at 3 atmospheres pressure is inspired, the amount of dissolved oxygen in the plasma increases to 6 ml./100 ml. (at a partial pressure of about 2000 mm. Hg).

At higher pressures proportionately greater amounts of oxygen are forced into solution. It is thus easy to understand how laboratory animals can be kept alive on erythrocyte-free blood substitutes under 4 or more atmospheres pressure breathing 100 per cent oxygen ("life without blood").[9]

Alterations in Gas Transport and Vasoconstriction

It has been demonstrated by Lambertsen and his group[16] that profound changes occur in blood-gas transport in the cerebral circulation of healthy volunteers subjected to hyperbaric oxygenation. It was shown that inspiration of oxygen under increased pressures leads to intense cerebral vasoconstriction. In addition, it was found that interference with the normal reduction of oxyhemoglobin causes a change in the transport of carbon dioxide. It was observed that the partial pressure of carbon dioxide of internal jugular venous blood rose about 3 mm. Hg at a pressure of 3½ atmospheres.

That the cerebral blood flow is indeed reduced by hyperbaric oxygen was corroborated by krypton-85 clearance studies of Bloor et al.[7] who showed that inspiration of oxygen at two atmospheres leads to a reduction of blood flow through the cerebral cortex of 21 per cent, associated with a reduction in oxygen consumption by the brain cells of 38 per cent.

Similarly it was shown by Bird and Telfer[6] that hyperbaric oxygen (two atmospheres absolute) causes a diminution of blood flow in the forearm of healthy young volunteers, with the result that the quantity of oxygen supplied to the forearm remains essentially unchanged. It should not necessarily be assumed that a like result would occur in an abnormal situation, for instance in arteriosclerotic occlusive disease or in traumatic arterial occlusion.

In another area, namely the pulmonary circulation, Moulder and his co-workers[18] have demonstrated the hypertensive and usually vasoconstrictive action of hyperoxia on the pulmonary vascular bed.

Thus, it has been recorded that increased oxygen tension in the blood can cause vasoconstriction in various organs. Bean[2] states, however, that at high pressures, such as above 4 atmospheres, there is a significant elevation in brain tissue oxygen in spite of cerebral vasoconstriction.

Much further investigation of the physiological action of hyperbaric oxygen is necessary before some of the apparent discrepancies between laboratory data and actual clinical practice are resolved.

SURGICAL APPLICATIONS

Protection During Induced Circulatory Arrest

Hyperbaric oxygenation may prove a valuable addition to methods already available which allow the surgeon to interrupt circulation to vital organs. Along with the heart-lung bypass and hypothermia, hyperbaric oxygenation ranks as an alternative protective device.

In the search for improved methods that would permit the surgeon to arrest circulation, hypothermia was introduced by Bigelow in Canada and Boerema in the Netherlands in 1949. Although hypothermia prolongs the period during which the surgeon is able to safely interrupt the circulation, it does not allow sufficient time to repair malformed or scarred valves or intracardiac defects meticulously. Furthermore, lowering of the body temperature below 28° C. increases the risk of ventricular fibrillation. With the development of the heart-lung bypass apparatus, many of these problems were solved, and the use of hypothermia was relegated to a more limited area.

Yet, the cardiopulmonary bypass is not without its drawbacks. Some of its disadvantages as well as those of hypothermia may be avoided in select instances by the use of hyperbaric oxygen instead. Thus, the experience of Bernhard and his co-workers suggests the value of hyperbaric oxygen in treating infants and small children with congenital heart disease when it is desirable to perform surgery without the use of a pump oxygenator.[3, 4] These workers believe that hyperbaric oxygenation provides sufficient protection for the infant with congenital cyanotic heart disease to permit surgery without the use of the cardiopulmonary bypass.

Soon after the introduction of hyperbaric oxygen in surgical research, it was found to be relatively simple to raise the oxygen content of the peripheral arterial blood of patients with congenital right-to-left shunts. With the use of hyperbaric oxygen the blood that courses through the pulmonary vascular bed is supplied with more than the normal volume of oxygen. When this blood mixes with the venous blood that enters the arterial circulation, its surplus of oxygen serves to oxygenate the venous blood. This mechanism was utilized by Bernhard, Somers, Krick and Abe[3] during corrective and palliative surgical procedures carried out on newborn blue babies in the hyperbaric chamber. Many of these infants are considered too small to be placed on the heart-lung bypass. Inflow occlusion is frequently used in these patients. Under hyperbaric oxygen,

Figure 1. View of the front portion of the hyperbaric unit at Lutheran General Hospital showing the entrances to the three chambers connected by intervening locks. The center one, having the widest diameter (12 ft.), is the surgical chamber shown in Figure 2.

inflow occlusion seems to be better tolerated. At times pressures as high as 3 and 4 atmospheres are used.

For example, creation of a systemic-pulmonary arterial shunt is often performed in very cyanotic patients with severely decreased pulmonary flow, as in pulmonary atresia, tetralogy of Fallot, tricuspid atresia and transposition of great vessels with pulmonary stenosis. These patients usually have a very low resting arterial pO_2 in spite of administration of oxygen by mask. While the anastomosis is being performed, one pulmonary artery is temporarily occluded, leading to further desaturation of the arterial blood. Many of these infants develop arrythmias and cardiac arrest. When hyperbaric oxygenation is used, the pO_2 of arterial blood is raised and the temporary occlusion is more easily tolerated.

Bernhard was recently reported to have compared one group containing 18 deaths in a series of 86 critically ill infants operated upon in the hyperbaric chamber with another group containing 18 deaths out of 56 similar patients operated on prior to the use of the chamber. This comparison would suggest optimism in regard to the use of the chamber. However, breakdown of these figures apparently revealed that the mortality rate for the cyanotic patient with tetralogy of Fallot treated in the chamber was 32 per cent, allegedly higher than the figure usually achieved without hyperbaric oxygenation.[17] The results of cardiac surgery conducted under increased atmospheric pressure will require more analysis before conclusions can be made.

Combining hyperbaric oxygen with the cardiopulmonary bypass has been offered as a method of improving the efficiency of the bypass and allowing lower perfusion rates. Closure of ventricular septal defects, replacement of valves and other intracardiac procedures require total cardiopulmonary bypass. During such operations, metabolic acidosis due to hypoxia of the tissues is avoided by using a relatively high perfusion flow (100 ml./kg. of body weight per minute). In some patients the required high perfusion rate is not easily achieved. The use of hyperbaric oxygenation in conjunction with low flow perfusion may prevent this metabolic imbalance.

Combining hyperbaric oxygen with hypothermia may offer additional advantages to the surgeon. Richards, Pinto and Coombs,[21] in studying the combined use of hypothermia and hyperbaric oxygenation, describe the prolongation of circulatory occlusion time which can be safely achieved. They observed that dogs could survive complete circulatory and respiratory occlusion for only three minutes at normal temperature and pressure. However, they could tolerate this occlusion for 45 to 60 minutes under combined hypothermia (28° to 30° C.) and hyperbaric oxygenation (3 atmospheres absolute, breathing 98 per cent O_2 and 2 per cent CO_2). They suggest that this type of suspended animation would be useful in some cardiovascular and neurosurgical operations.

Figure 2. Surgical chamber of the hyperbaric unit at Lutheran General Hospital as viewed through the door of the adjacent lock.

An interesting finding has been reported by Wang, Lesko and Jacobson[24] in their observation that certain stress situations are better borne when surgery is performed under hyperbaric oxygenation. For instance, removal of the occluding clamp from the abdominal aorta following aortic surgery can be performed with less hazard and less hypotensive response when the patient is under hyperbaric oxygenation. This clinical observation seems to be substantiated by their experiments from which they conclude that "hyperbaric oxygenation at 3 atmospheres absolute maintains the resting tissue oxygen requirements following aortic occlusion in dogs."

Such a conclusion correlates well with the observations of Schwartz and Breslau,[22] who occluded the thoracic aorta in dogs and found that 100 per cent oxygen at 4 atmospheres pressure protected the animals from developing the spinal cord and visceral damage which usually follows thoracic aortic occlusion.

Our experience during aortic resection and graft replacement performed in 4 patients under hyperbaric oxygenation is similar to that of Jacobson and co-workers in that removal of the aortic occluding clamp was not followed by the usual drop in blood pressure, nor was it necessary to remove the clamp very slowly as is customary. Our operative experience further correlates with Jacobson's[15] in that we believe "low-risk" surgical patients (e.g., patients with a history of myocardial infarction) might enjoy additional protection during their operation if it were performed under hyperbaric oxygenation. There is as yet, however, no clinical evidence to support our opinion.

Homotransplantation

Recent studies demonstrate that hyperbaric oxygenation seems useful in the procurement of cadaver kidneys prior to homografting. Hitchcock and Haglin[12] reported the combination of hyperbaric oxygenation, mild hypothermia and a method of mechanically propelling blood in the cadaver, thus maintaining blood flow in the distal aorta prior to the removal of the kidneys. They report pO_2 values of over 1800 mm. Hg in the distal aorta using 100 per cent oxygen at 3 atmospheres pressure. This method has allowed more time for the procurement of these donor kidneys in preparation for homotransplantation. Prolonged ischemia times up to three and one-half hours were reported with early function of the kidneys in five to six hours. Similar techniques should be useful in transplanting other organs as well.

Clostridial Infection

Perhaps the most encouraging results of hyperbaric oxygen exposure

have been obtained in the treatment of patients with gas gangrene.[10] When Boerema was faced with the management of two severely ill patients with gas gangrene in whom amputation of the affected limb was not possible, it was only logical to extend his research work to the treatment of anaerobic infections. These two patients were treated in his high pressure chamber with oxygen at 3 atmospheres of pressure for two hours at a time, three times a day. The advance of the gas gangrene was dramatically arrested, and the systemic toxicity disappeared rapidly. Subsequently a series of over 50 patients with Clostridium perfringens infections were treated in his chamber with considerable success; the usual high mortality rate was greatly reduced and frequently amputation was avoided.

Thus far a series of 22 patients with gas gangrene has been treated in the pressure chambers of Lutheran General Hospital with a similar good response. It is difficult to compare this new form of therapy with established methods. Evaluation is all the more difficult since we found that hyperbaric oxygen is not bactericidal to Clostridium perfringens (welchii), as the infecting organism can be cultured from the wounds following clinical improvement of the patient.

A rather dramatic example was a 36 year old man who had developed a large soft-tissue slough of the right gluteal area following an automobile accident. The wound had been debrided immediately, but on the third day his temperature rose rapidly, and on the morning of the fourth day a diagnosis of anaerobic myositis and cellulitis was made. The infectious process involved the right buttock and the right flank and extended over the anterior aspect of the abdomen to the left anterosuperior spine, and down the lateral aspect of the right thigh as manifested by bronzing of the skin and crepitation. He was semicomatose. The heart rate was 150 per minute, and the temperature was 102° F. Forty-five minutes following the beginning of hyperbaric oxygen therapy he became more alert and manifested an interest in his surroundings. No progression of the disease was noted thereafter. On the contrary, he improved rapidly. No gas gangrene antitoxin was given. Despite a rapid clinical improvement cultures from the wound continued to show Clostridium perfringens (welchii) up to 10 days following the start of therapy. On the tenth day, the patient was returned to his home town with healthy granulation tissue covering the wound and in generally good condition, and following plastic repair he was able to return to work.

Following the encouraging results in the treatment of severe gas gangrene, high pressure oxygen was also applied to patients with tetanus. Despite enthusiastic reports from other centers, two patients with tetanus were treated in the hyperbaric oxygen chamber of Lutheran General Hospital without any favorable response and both patients died.

Acute Arterial Insufficiency

Sufficient clinical observations have now been made to show that certain types of acute arterial insufficiency, such as that resulting from trauma, may receive benefit from hyperbaric oxygenation. A few cases have been described in which an ischemic limb has been salvaged by hyperbaric oxygen therapy following severe injury to peripherally located small arteries, often in association with fractures and damaged soft tissues. Another variety of acute arterial insufficiency that may respond favorably to hyperbaric oxygen therapy is found in patients with severe vasospasm developing as a toxic reaction to certain drugs. Frostbite is still another variety of acute vascular insufficiency that seems to respond well to hyperbaric oxygen. In all probability, the main function of the hyperbaric oxygen chamber in these cases of acute arterial insufficiency is maintenance of viability of the extremity until secondary vasospasm diminishes and collaterals develop.

A 25 year old man was treated in our chamber for vascular insufficiency of the right foot due to trauma to the leg. The foot was cyanotic and cold and did not seem viable. With exposure to hyperbaric oxygen the bluish discoloration of the foot changed initially only very little. However, a bordering zone of pink color soon began to invade the blue area and enlarging islands of pink color appeared within the blue area. Ultimately amputation of the foot was avoided, although dry gangrene of the terminal phalanx of the third and fourth toes developed.

Hyperbaric oxygen was also incorporated in the successful treatment of severe frostbite of the feet of two boys (12 and 13 years old). Frostbite had developed during exposure to cold temperature while the shoes had been laced too tightly. The toes and the distal portion of the feet were deeply cyanotic and did not seem viable. With hyperbaric oxygen the color of the toes turned pink, although initially they returned to a gray-blue discoloration upon removal of the patient from the hyperbaric oxygen environment. Following subsequent treatments normal color was regained and viability established. Admittedly, it is difficult to evaluate the true role of hyperbaric oxygen in such cases without a control series.

Chronic Arterial Insufficiency

In *chronic* arterial occlusive disease, unfortunately, prolonged benefit from hyperbaric oxygen therapy has not been observed. Although Boerema has reported a favorable response, the experience at our institution with this disease is that hyperbaric oxygen is of little or no value. In a series of more than 50 patients with advanced arteriosclerotic occlusive disease with severe peripheral ischemia subjected to prolonged courses of therapy in the hyperbaric oxygen chamber at Lutheran General Hospital, none

has shown significant change. Occasional subjective improvement is experienced by some of these patients while actually inside the hyperbaric chamber, only to be followed by a return to prior status on leaving the chamber and returning to the normal environment. This could very well be a placebo effect.

Hypovolemic Shock

Interesting studies have been reported by Attar and co-workers,[1] who found that oxygen at a pressure of 3 atmospheres improves significantly the survival rate of dogs in which hemorrhagic shock had been produced. A similar degree of improvement of the survival rate was found in traumatic shock (produced in rats in a Noble-Collip drum). Dogs in bacteremic shock failed to show any response to exposure to high pressure oxygen.

Yet it has been observed by Nora and Laufman that experimentally induced strangulated obstruction of the intestine (in rats) has been followed by an increase in survival rates, when the animal is subjected to hyperbaric oxygen soon after the obstruction is induced.[19]

Laboratory Research

A most important function of the hyperbaric chamber at the present time is its use as a research device. There are many questions which must be answered, such as how to best counteract the vasoconstrictive property of hyperbaric oxygen (e.g., using CO_2), which at times threatens to neutralize the beneficial action of the chamber.

Considerable information on oxygen toxicity has accumulated over the past several decades, thanks to those investigators concerned with the physiology of diving and the submarine environment. This wide subject has been extensively treated elsewhere and is considered beyond the scope of this paper. It, too, is a field in which more study is indicated.

A particularly important area of challenge is that having to do with the problem of myocardial ischemia, and the possible value of hyperbaric oxygen in treating this disease has been considered by many. In our laboratory a project is in progress attempting to evaluate the effect of hyperbaric oxygen in acute ischemic heart disease. Myocardial infarction is produced in dogs by occlusion of the left circumflex artery[23] and also by occlusion of both the left circumflex and anterior descending arteries. Thus far a preliminary conclusion can be made that high pressure oxygen inhibits the development of ventricular fibrillation resulting from these acute occlusions. Illingworth,[13] Petropoulos,[20] and numerous others have embarked upon this investigation and have found similar protective action. Further study is indicated to determine the possible protective

value of hyperbaric oxygen during surgical revascularization of the myocardium.

Hyperbaric oxygenation has also been suggested for the treatment of patients with massive pulmonary emboli and as an adjuvant to surgical attempts to remove such emboli.

The potential areas for investigation of hyperbaric oxygen are legion. Recent overenthusiasm for hyperbaric oxygen as a clinical panacea has subsided, and current interest is acquiring a healthy attitude of cautious acceptance. It is hoped that a mature perspective will be attained during the ensuing years of continuing research and additional clinical experience.

REFERENCES

1. Attar, S., Esmond, W. G. and Cowley, R. A.: Hyperbaric oxygenation in vascular collapse. J. Thoracic & Cardiovasc. Surg. *44:* 759, 1962.
2. Bean, J. W.: Background of clinical hyperbaric oxygenation. Proc. Workshop on Hyperbaric Oxygen Research of Lutheran General Hospital, February 5, 1964.
3. Bernhard, W. F., Somers, L. A., Kriek, H. R. and Abe, T.: Corrective and palliative surgery in infants and children with congenital heart disease. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 189.
4. Bernhard, W. F. and Tank, E. S.: Effect of oxygen inhalation at 3.0 to 3.6 atmospheres absolute upon infants with cyanotic congenital heart disease. Surgery *54:* 203, 1963.
5. Bert, P.: La Pression barometrique, recherches de physiologie experimentale. Paris, G. Masson, 1878.
6. Bird, A. D. and Telfer, A. B. M.: Effect of hyperbaric oxygen on limb circulation. Lancet *1:* 355–356 (Feb. 13) 1965.
7. Bloor, K., Bratten, N. T., Jacobson, I., McCaffrey, J. F. and McDowall, D. G.: Low flow perfusion with total heart-lung bypass at two atmospheres absolute pressure. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 194.
8. Boerema, I.: An operating room with high atmospheric pressure. Surgery *49:* 291, 1961.
9. Boerema, I., Meijne, N. G., Brummelkamp, W. K., Bouma, S., Mensch, M. H., Kamermans, F., Hanf, M. S. and Van Aalderen, W.: Life without blood. J. Thoracic & Cardiovasc. Surg. *1:* 133, 1960.
10. Brummelkamp, W. H.: Treatment of infections with Clostridium welchii by oxygen therapy at 3 atmospheres. A report on 37 cases. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 20.
11. Brummelkamp, W. H., Boerema, I. and Hoogendijk, J. L.: Treatment of anaerobic infection (clostridial myositis) by observing tissues with oxygen under high pressure. Surgery *49:* 299, 1961.
12. Hitchcock, C. R. and Haglin, J. J.: Present status of hyperbaric oxygenation in surgery. Presented at the Twenty-Second Annual Meeting of the Central Surgical Association, Milwaukee, Wisconsin, 1965. To be published.
13. Illingworth, C.: Treatment of arterial occlusion under oxygen at two-atmospheres pressure. Brit. M. J. *5315:* 1271–1275 (Nov. 17), 1962.

14. Jacobson, J. H., II, Morsch, J. H. C. and Rendell-Baker, L.: The historical perspective of hyperbaric therapy. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 7.
15. Jacobson, J. H., II: Personal communication.
16. Lambertsen, C. J., Kough, R. H., Cooper, D. Y., Emmel, G. L., Loeschcke, H. H. and Schmidt, C. F.: Oxygen toxicity; effects in man of oxygen inhalation at 1 and 3.5 atmospheres upon blood gas transport, cerebral circulation and cerebral metabolism. J. Appl. Physiol. *5:* 471, 1953.
17. Maloney, J. V.: Cardiovascular surgery. Surg. Gynec. & Obst. *120:* 268, 1965.
18. Moulder, P. V., Lancaster, J. R., Harrison, R. W., Michel, S. L., Snyder, M. and Thompson, R. G.: Pulmonary arterial hyperoxia producing increased pulmonary vascular resistance. J. Thoracic & Cardiovasc. Surg. *40:* 588, 1960.
19. Nora, P. and Laufman, H.: Personal communication.
20. Petropoulos, P. C.: Influence of hyperbaric oxygenation on the haemodynamic changes and mortality after circumflex coronary artery occlusion. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 84.
21. Richards, V., Pinto, D. and Coombs, P.: Considerations and uses of hyperbaric oxygen therapy in surgery. Am. J. Surg. *106:* 114, 1963.
22. Schwartz, S. and Breslau, R. C.: Protective effect of hyperbaric oxygenation during thoracic aortic occlusion. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 395.
23. Van Elk, J. and Trippel, O. H.: Ventricular fibrillation following coronary occlusion in the dog heart and the possible protective effect of high pressure oxygen. *In* Boerema, I., Brummelkamp, W. H. and Meijne, N. G.: Clinical Application of Hyperbaric Oxygen. Amsterdam, Elsevier Publishing Co., 1964, p. 116.
24. Wang, M. C. H., Lesko, W. S. and Jacobson, J. H.: Hyperbaric oxygenation: Oxygen exchange in an acutely ischemic vascular bed. Presented at the Nineteenth Annual Meeting of the Society for Vascular Surgery, New York City, June 20, 1965. To be published.
25. Wangensteen, O. H.: Intestinal Obstruction. 3rd Ed. Springfield, Charles C Thomas, 1955, p. 190.

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ADDITIONAL ARTICLE

Technique of Single-Layer End-to-End Intestinal Anastomosis by Triangulation

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Achievement of an intestinal anastomosis that is functionally and anatomically sound depends above all on the maintenance of unimpaired blood supply to the anastomotic ends and on the coaptation of these ends without tension. When these conditions are met, the attention of the surgeon can then be directed to good suture approximation and to preservation of a lumen of adequate caliber. Although mechanically the strongest union can be obtained by multilayer closure with continuous, nonabsorbable sutures, this procedure tends to infold so much tissue that the lumen may be unduly narrowed. An anastomosis to prevent narrowing of the lumen could be achieved by exact approximation of all the layers of the cut intestinal ends; however, exact approximation with sutures is not practical, and may even be impossible, but such an approximation may some day be realized with the development and refinement of stapling devices. Another method of preventing narrowing of the lumen, namely suturing of the bowel ends so as to evert them, has often produced good healing in experimental animals.[4] However, instances of suture line leakage and adhesions to other bowel loops have occurred, so that this method has not been recommended for the human subject.

The problem that the surgeon must solve, in the present state of technical development, is to invert enough tissue with sutures to maintain a leak-proof anastomosis, but not enough to narrow the lumen exces-

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sively. It is the purpose of this paper to describe a method of solving this problem.

The technique utilizes the principle of triangulation of the transected ends, a method often employed for blood vessel anastomosis. It is similar to the two-layer technique previously described,[5] but uses only a single layer of seromuscular sutures. Abandonment of the two-layer in favor of the one-layer technique is based on sound theoretical and practical considerations. A continuous catgut layer, usually recommended for the inner layer of a two-layer closure, narrows the lumen by its purse-string effect. Two layers of sutures fold into the lumen more tissue than one layer. Animal experiments by Buchin and Van Geertruyden[2] showed more tissue necrosis and microscopic abscesses in two-layer than in one-layer anastomoses. Clinically, the same authors described significantly more complications, morbidity and mortality with a double than with a single layer technique.

I recently compared the results on 50 patients whose intestinal continuity had been restored by the two-layer triangulation technique with those of 50 patients whose anastomoses were performed by the single layer technique.[6] Although the mortality and late complication rates were not different in the two groups, gas and stool passage occurred on the average one day earlier with the one-layer technique. One-layer closures have been used successfully by many surgeons, both here and abroad, notably Gambee et al.,[3] Jourdan,[7] and Beling.[1]

The technique to be described requires delicacy and finesse, and is not for the surgeon whose primary consideration is speed. Tissues are handled with gentleness, using small nontraumatizing instruments. Interrupted sutures of 30–35 cm. lengths of 4-0 braided silk on one-half circle No. 16 Ferguson needles have proved most useful for the anastomosis. Inherent in the technique is the ability to overcome disparities in the calibers of the lumens and to recognize the adequacy of blood supply to the anastomotic ends. The technique is equally useful for small and large bowel anastomoses. By careful attention to detail, an anastomosis satisfying the requirements set forth above, as well as the surgeon's esthetic sense, can be obtained.

OPERATIVE TECHNIQUE

Before the bowel is resected, adequate mobilization should be obtained so that the ends can be approximated without tension. This is seldom a problem with the small intestine or after right colectomy, but after left colectomy often requires mobilizing the splenic flexure. The resection planes are chosen, and a small opening is made in the mesentery imme-

diately adjacent to the bowel, one for each resection plane (Fig. 1, *A*). A straight crushing clamp is applied across the bowel with the tip barely extending into this opening. The angle at which the clamp is placed with reference to the long axis of the bowel is of some importance, and may vary from 90 to 45 degrees, depending upon how much disparity exists between the lumen calibers of the afferent and efferent ends. The more acute the angle, the larger the diameter of the lumen. However, a reasonable difference in the size of the lumens can be overcome by the triangulation technique.

The amount of mesentery to be resected will, of course, depend on the pathologic process. It is best to visualize the mesenteric vessels in transmitted light, so that they can be individually ligated and transected, thereby avoiding massive transfixion ligatures of the cut mesentery (Fig. 1, *B*). The bowel proximal to the proximal clamp and distal to the distal clamp is carefully cleared of mesentery and of all adventitious tissue for a distance of 1.0 to 1.5 cm. This requires division and ligation of each small mesenteric vessel at the bowel, and removal of the appendices epiploicae of the large intestine. The end result of this clearance is a cylinder of bowel 1.0 to 1.5 cm. long whose seromuscular exterior is completely exposed and ready for suturing. To prevent soilage, the afferent loop of bowel is milked of its contents and a rubber- or mesh-shod clamp is applied to prevent it from refilling (Fig. 1, *B*). Often, the bowel has been so well prepared that this is unnecessary. In most instances, the efferent loop does not require emptying or clamping. Hugging the proximal side of the proximal clamp and the distal side of the distal clamp, the intestine is divided starting at the antimesenteric border. As the division proceeds, the individual bleeders, located almost entirely in the submucosal layer, are clamped with mosquito clamps and tied with 5-0 silk. If there is arterial bleeding, satisfactory vascularity of the ends of the intestine to be joined is assured. Moreover, that portion of the intestine lying in the jaws of the crushing clamp is removed with the specimen.

After removal of the diseased intestine, the nubbins of mesentery closest to the intestine are sutured together. If this can be done without tension, the chances are there will be little pull on the anastomosis. If there is too much tension, additional mobilization may be required. The actual joining of the ends of intestine is performed by the careful application of seromuscular Lembert sutures. These sutures are placed in the cuffs of tissue previously cleared. The first suture, guide suture *a*, joins the cleared mesenteric borders and is knotted and tagged. Two more seromuscular guide sutures, *b* and *c*, are applied, each a third the circumferential distance from *a* and from each other. These, too, are tied and tagged (Fig. 2, *C*). The circumference of the bowel is thereby triangulated (Fig. 2, *D*).

Figure 1. Technique of single-layer end-to-end intestinal anastomosis by triangulation.

A, Openings are made in a vascular site in the mesentery immediately bordering the chosen resection planes, and crushing straight clamps are placed across the bowel, the points barely extending into the openings. The clamps are angulated toward the long axis of the bowel to increase the circumference of the cut ends, and to remove more of the antimesenteric than of the mesenteric border. The dotted lines indicate resection line in the mesentery. The illustrations show large intestine, but the technique is applicable to the small intestine as well.

B, The mesenteric blood vessels are visualized in transmitted light and are individually isolated, clamped and ligated, following which the mesentery is transected. A rubber-shod non-crushing clamp is applied to control fecal flow from the afferent loop. The mesenteric half of the afferent loop is divided with scalpel hugging the proximal side of the crushing clamp. In practice, hemostasis of the cut end is more easily obtained if transection begins on the antimesenteric side. The division progresses a little at a time, so that the individual bleeding points, most of which are in the submucosa, can be secured with mosquito clamps and tied with 5–0 silk. The afferent loop is similarly divided along the distal side of the distal crushing clamp. (*Continued in Figure 2.*)

(*Conclusion of legend to Figure 2*)

1. By opposing pull on guide sutures *b* and *c,* the third of the circumference lying between them is closed with interrupted seromuscular sutures.

2. The third of the circumferential ends between sutures *a* and *b* is approximated with similarly placed seromuscular sutures.

3. Guide suture *a* is carried under the bowel. Opposing pull on suture *c* brings into relief the circumference lying between *a* and *c,* which is then similarly closed.

E, The rubber-shod clamp is removed. The suture line is inspected and reinforced if needed. The guide sutures are cut and the mesenteric defect is closed.

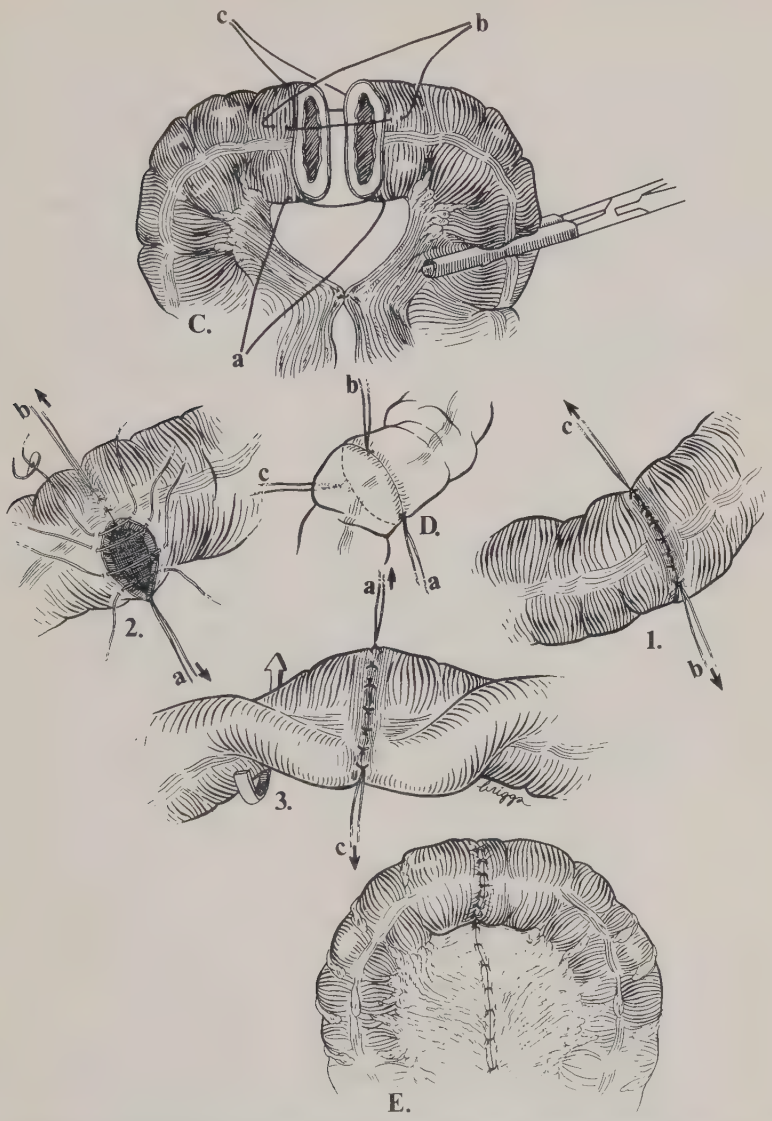

Figure 2. Technique of single-layer intestinal end-to-end anastomosis by triangulation (*continued*).

C, The bowel is cleared of mesentery, the appendices epiploicae and adventitious tissue for a distance of 1.0 to 1.5 cm. from the cut ends. The mesenteric nubbins adjacent to the intestine are approximated. The cleared mesenteric borders of the bowel are approximated by a seromuscular Lembert suture of 4–0 silk, guide suture *a*. Two more seromuscular Lembert sutures, guide sutures *b* and *c*, are similarly placed, each a third the circumferential distance from suture *a* and from each other.

D, Guide sutures *a*, *b* and *c* are tied and tagged, thereby triangulating the circumference of the approximated ends.

(*Legend concluded on opposite page*)

Now the third of the circumference lying between guide sutures *b* and *c* is brought into relief by pulling them in opposite directions at right angle to the bowel. The ends of this third are then coaptated with interrupted sutures which are tied and cut (Fig. 2, *D*, 1). The third lying between guide sutures *a* and *b* is similarly sutured (Fig. 2, *D*, 2). In order to put into relief the third of the circumference lying between guide sutures *a* and *c*, suture *a* must be passed between the bowel and the joined mesenteric nubbins (Fig. 2, *D*, 3). Between four and seven sutures are usually required to approximate the ends of each third of the circumference. The clamp applied for control of the fecal stream is removed. After inspection of the suture line and reinforcement, if required, the guide sutures are cut. The mesenteric defect is closed, thereby completing the anastomosis (Fig. 2, *E*).

SUMMARY

A detailed technique of open end-to-end intestinal anastomosis is described and illustrated. The method features triangulation of the divided ends, and the careful application of interrupted seromuscular Lembert sutures of 4-0 silk in a single layer. Inherent in the technique is the exclusion of crushed tissue from the anastomosis, the ability to observe the adequacy of blood supply to the cut ends, and the capability of overcoming disparities in the lumens of the afferent and efferent loops. Its proper execution insures solid union without compromising the lumen.

REFERENCES

1. Beling, C. A.: Single layer end-to-end intestinal anastomosis. Am. J. Gastroenterol. *27:* 374–379 (April) 1957.
2. Buchin, R. and Van Geertruyden, J.: Valeur comparee des sutures intestinales en un plan et en deux plans. Acta chir. belg. *50:* 461–480 (June) 1960.
3. Gambee, L. D., Garnjobst, W., and Hardwick, C. E.: Ten years' experience with a single layer anastomosis in colon surgery. Am. J. Surg. *92:* 222–225 (Aug.) 1956.
4. Healey, J. E., McBride, C. M. and Gallager, H. S.: Is serosa-to-serosa approximation necessary in intestinal anastomosis? S. Forum *15:* 297–299, 1964.
5. Heifetz, C. J.: A Technique for open end-to-end intestinal anastomosis. S. Clin. North America *30:* 1481–1490 (Oct.) 1950.
6. Heifetz, C. J.: One-layer versus two-layer intestinal anastomosis: Clinical study. Tr. 14th Biennial International Congress, International College of Surgeons, 555–562, May, 1964.
7. Jourdan, P.: A propos de la suture a plan unique des tuniques digestives. Acta chir. belg. *54:* 765–771 (Nov.) 1955.

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Index

NOTE: Page numbers of symposium and clinic titles are in **boldface** type.

ACOUSTIC neuroma, diagnosis and surgical treatment, 127–129
Anastomosis, intestinal, by triangulation, single-layer end-to-end, technique, **223–228**
Anesthesia, for bronchopulmonary sleeve resection after preoperative irradiation for cancer, 146
for emergency portacaval shunt, 39
Angina, intestinal, surgery of, 171
Aorta, abdominal, branches of, surgery on, 170
Aortic valvular disease, surgery of, present status, 189
Aortoiliac occlusive disease, surgery of, 173
Arterial insufficiency, hyperbaric oxygen in, 218
Arteriography in cerebrovascular disease, 163
Artery(ies), small, diseases of, acquired, surgery of, **161–174**
Atherosclerosis, coronary, surgery for, 169
Audiometry in acoustic neuroma, 127
Axilla, dissection, anatomical considerations, 77
technique, **77–83**
Azathioprine for immune suppression in renal transplantation, 22

BILE ducts, stricture, due to operative injury, 73
malignant, 74
Biliary tract, diseases, diagnostic methods, **69–76**
surgery of, present status, **69–76**
Brachiocephalic arterial repair, technique, 167
Breast, lymphatic drainage, 78
Bronchopulmonary sleeve resection after preoperative irradiation for lung cancer, **145–159**

CARCINOMA, of bile ducts, 74
of larynx, advanced, combined radiation and surgery in, 104
preservation of function by subtotal laryngectomy, 100
of lung, preoperative irradiation and bronchopulmonary sleeve resection for, **145–159**
of parotid gland, operation for, employing partial temporal bone resection, **131–143**
Cardiopulmonary bypass, hyperbaric oxygen with, 212
Carotid bypass graft techniques, 169
Carotid endarterectomy for stroke, technique, 167
Cerebrovascular disease, arteriography in, 163
clinical examination, 162
surgery of, 162–169
indications for, 165
Children, heart disease in, new operations, **175–187**
Cholangiography, intravenous, in diagnosis, 69
needle, indications and technique, 71
transhepatic, percutaneous, technique and principles, 70
Cholecystography, oral, in diagnosis, 69
Circulation, arrest of, induced, hyperbaric oxygen as protection during, 212
Claudication, intestinal, surgery of, 171
Clips, vena cava, 201, 202
Clostridial infections, hyperbaric oxygen in, 216
Coma, hepatic, pre- and postoperative, in emergency portacaval shunt, 52
Common duct, stricture, due to operative injury, 73
malignant, 74
Cordotomy, percutaneous, for pain, **3–12**
intramedullary, direct current, technique, 5–8

Cordotomy (*Continued*)
percutaneous, intramedullary, radiofrequency, technique, 8
strontium, technique, 4
Coronary artery disease, surgery of, present status, 192
Coronary atherosclerosis, surgery for, 169
Coronary endarterectomy, 170

DEAFNESS due to otosclerosis, surgery for, 112–118
Dissection of axilla, technique, **77–83**
Dizziness in Meniere's disease, relief of, 118
Duodenum, obstructing hematoma of, traumatic, operation for **85–97**
pathogenesis and clinical picture, 86, 87

EAR, microsurgery of, **111–130**
Ebstein's malformation, correction of, 183
Embolectomy, pulmonary, indications and technique, **27–36**
Embolism, pulmonary, diagnosis, 28
prevention, vena cava operations for, indications and technique, **195–207**
Endarterectomy, carotid, for stroke, technique, 167
coronary, 170
Endolymphatic sac, operations on, for Meniere's disease, 125
Esophagogastric varices, bleeding, diagnosis, 38
Extremity, lower, arterial disease, surgery of, 172
upper, lymphatic drainage, 78

FEMORAL artery, occlusive disease, surgery of, 173
Fenestration operation for otosclerosis, 113
Filter operation, vena cava, 201, 202, 204

GAS gangrene, hyperbaric oxygen in, 216
Glottic rehabilitation in vocal cord paralysis, 99
Goldblatt phenomenon, surgical relief, 170

HARRINGTON'S method of instrumentation in treatment of scoliosis, 57
Heart, block, surgery of, present status, 191
disease, acquired, surgery of, **189–193**
in children, new operations in **175–187**
Hematoma, traumatic, obstructing, of duodenum, operation for **85–97**
pathogenesis and clinical picture, 86, 87
Hemorrhage, postoperative, in emergency portacaval shunt, 51
Homotransplantation, hyperbaric oxygen in, 216
renal, **13–25**
Hyperbaric oxygen. See *Oxygen, hyperbaric.*
Hypertension, pulmonary, in septal defect, right pulmonary artery banding in, 176
renovascular, surgery of, 170
Hypothermia, hyperbaric oxygen with, 214

IMMUNE suppression in renal transplantation, 22
Imuran. See *Azathioprine.*
Infections, anaerobic, treatment with hyperbaric oxygen, 216
Instrumentation, internal, in treatment of scoliosis, **55–67**
Intestines, anastomosis, by triangulation, single-layer, end-to-end, technique, **223–228**
ischemia, surgery for, 170
Intraperitoneal small vessel surgery, 170
Ischemia, intestinal, surgery for, 170

KIDNEY, transplantation of, **13–25**
immune suppression in, 22
operative technique, 15
team approach, 15

LABYRINTHECTOMY for Meniere's disease, 124
Labyrinthotomy for Meniere's disease, 124
Laryngectomy, subtotal, for cancer, laryngeal function conserved by, 100
Larynx, carcinoma, advanced, combined radiation and surgery in, 104
preservation of function by subtotal laryngectomy, 100
surgery of, recent trends, **99–109**
trauma, 106
Ligation of vena cava, 201, 202, 203

Lung, carcinoma, preoperative irradiation and bronchopulmonary sleeve resection for, **145–159**
Lymph nodes, axillary, 78

MENIERE'S disease, symptoms and histopathology, 118, 120
surgery for, 118–126
ultrasonic therapy for, 126
Microsurgery of ear, **111–130**
Mitral valvular insufficiency, surgery of, present status, 190
Mitral stenosis, surgery of, present status, 189

NEPHRECTOMY, recipient, in renal transplantation, 20
Neurologic examination in acoustic neuroma, 128
Neuroma, acoustic, diagnosis and surgical treatment, 127–129

OPERATIONS, new, symposium on, **1–228**
Otosclerosis, surgery for, 112–118
Oxygen, hyperbaric, as protection during induced circulatory arrest, 212
in anaerobic infections, 216
in arterial insufficiency, acute and chronic, 218
in homotransplantation, 216
in hypovolemic shock, 219
physiologic considerations, 210
surgical uses, **209–221**

PAIN, percutaneous cordotomy for, **3–12**
Paralysis, vocal cord, glottic rehabilitation in, 99
Parotid gland, carcinoma, operation for, employing partial temporal bone resection, **131–143**
Peritoneum, small vessels of, surgery on, 170
Plication, vena cava, 201, 202, 203
Portacaval shunt, emergency, postoperative care and complications, 49
technique, **37–53**
Profunda revascularization in occlusive arterial disease, 174
Pulmonary artery, atresia, with normal aortic root, new operation for, 181
right, banding of, in septal defect with pulmonary hypertension, 176
Pulmonary embolectomy, indications and technique, **27–36**
Pulmonary embolism, prevention, vena cava operations for, indications and technique, **195–207**

RADIATION with surgery for advanced laryngeal cancer, 104
Renal artery, obstructions, surgery of, 170
Roentgen treatment, preoperative, of lung cancer, bronchopulmonary sleeve resection after, **145–159**

SACCULE, surgical rupture of, in Meniere's disease, 126
Scoliosis, treatment, internal instrumentation in, **55–67**
Harrington's method and review of cases, 57
Septal defects, with pulmonary hypertension, right pulmonary artery banding in, 176
Shock, hypovolemic, hyperbaric oxygen in, 219
Shunts, caval and systemic combined, in pulmonary atresia with normal aortic root, 181
Sleeve resection, bronchopulmonary, after preoperative irradiation for lung cancer, **145–159**
Splenectomy with renal transplantation, 20
Stapedectomy, total, for otosclerosis, 116
Stapes, mobilization, in otosclerosis, 114
Stricture, of common duct, due to operative injury, 73
malignant, 74
Stroke, acute, surgery of, 166
Strontium cordotomy, percutaneous, technique, 4

THYMECTOMY with renal transplantation, 20
Transposition of great vessels, complete, 176
Transplantation, of kidney, **13–25**
of tissues and organs, hyperbaric oxygen in, 216

Trauma, laryngeal, 106
Triangulation technique of single-layer, end-to-end intestinal anastomosis, **223–228**
Tricuspid atresia, correction of, total, 182
Trunk, upper, lymphatic drainage, 79

ULTRASONIC therapy for Meniere's disease, 126
Uremia, transplantation of kidney for, **13–25**
Ureter, anastomosis, in renal transplantation, 19

VALVES, cardiac, diseases of, status of surgery in, 189
prosthetic, 191
Varices, esophagogastric, bleeding, diagnosis, 38
Vena cava, clips, 201, 202
filter operation, 201, 202, 204
ligation, 201, 202, 203
operations on, to prevent pulmonary embolism, indications and technique, **195–207**
plication, 201, 202, 203
Vertebral subclavian steal syndrome, surgical treatment, 169
Vestibular function, total or partial destruction, for Meniere's disease, 124, 125
Vestibular nerve, intracranial, section of, for Meniere's disease, 125
Vestibular testing in acoustic neuroma, 127
Vocal cord, paralysis, glottic rehabilitation in, 99

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